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CONTENTS

THE ELECTROCARDIOGRAPH ELECTROLYTES, AND TOXEMIA OF PREGNANCY, *Willard F. Preston, M. D., Edward M. Bohan, M. D.*, and *J. Richard Durham, M. D.*, Wilmington, Del. 103

FURTHER OBSERVATIONS ON THE USE OF THE POST-PRANDIAL BLOOD SUGAR IN THE TREATMENT OF DIABETES MELLITUS, *Edward M. Bohan, M. D., Joseph L. Ackerman, M. D., Thomas V. Hynes, M. D.*, and *Harold A. Tarrant, M. D.*, Wilmington, Del. 108

SURGICAL CONSIDERATIONS OF THROMBO-EMBOLIC PHENOMENA, *Charles J. Dougherty, M. D.*, Wilmington, Del. 114

THE EARLY DIAGNOSIS OF POLIOMYELITIS, *George J. Boines, M. D.*, Wilmington, Del. 119

THE DELAWARE ACADEMY OF MEDICINE, *Lewis B. Flinn, M. D.*, Wilmington, Del. 121

EDITORIALS 125

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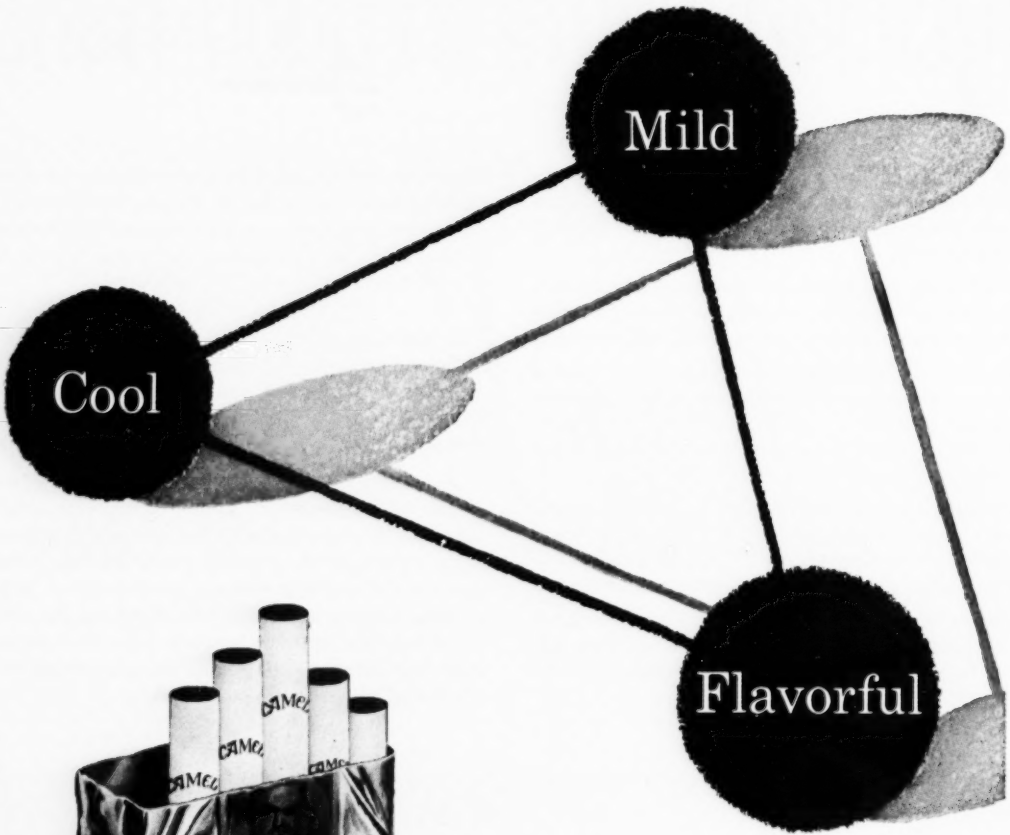

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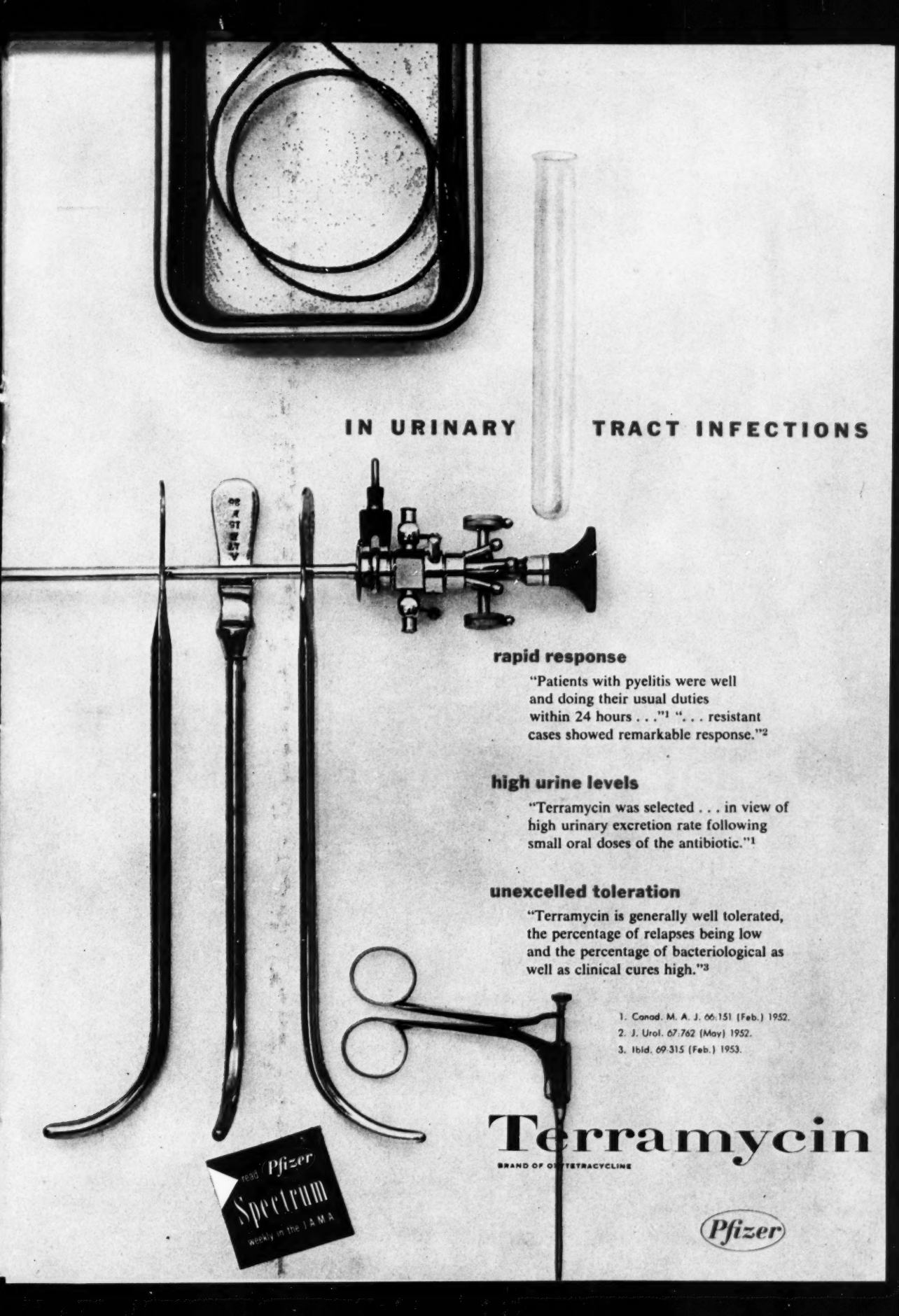

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1. Canad. M. A. J. 66:151 (Feb.) 1952.
2. J. Urol. 67:762 (May) 1952.
3. Ibid. 69:315 (Feb.) 1953.

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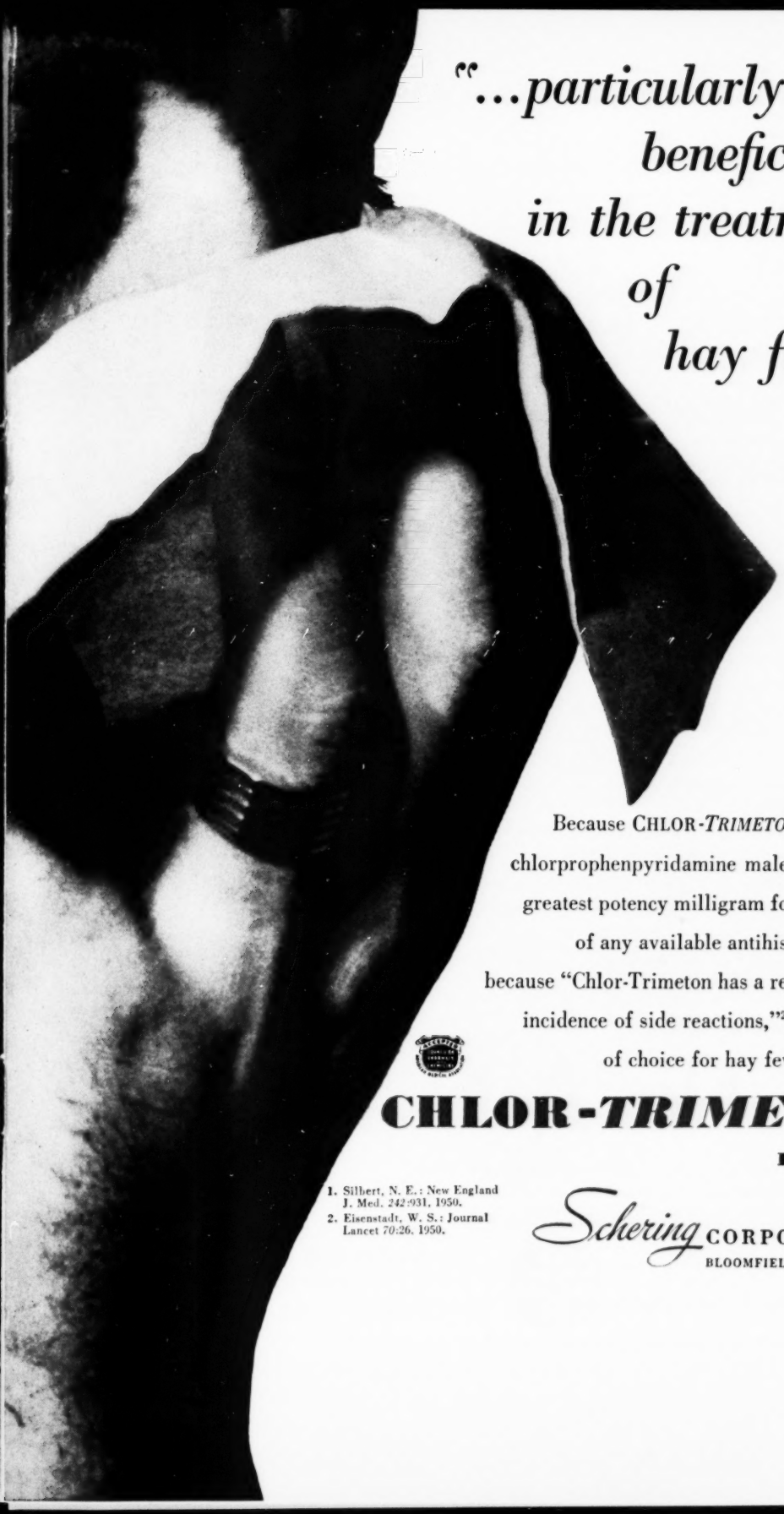

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1. Silbert, N. E.: New England J. Med. 242:931, 1950.
2. Eisenstadt, W. S.: Journal Lancet 70:26, 1950.

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*Food for Your Heart, a Manual for Patient and Physician, Department of Nutrition, Harvard School of Public Health, Harvard University, The American Heart Association, Inc., New York, 1952. Copies available through local Heart Association.

The Seal of Acceptance denotes that the nutritional statements made in this advertisement are acceptable to the Council on Foods and Nutrition of the American Medical Association.

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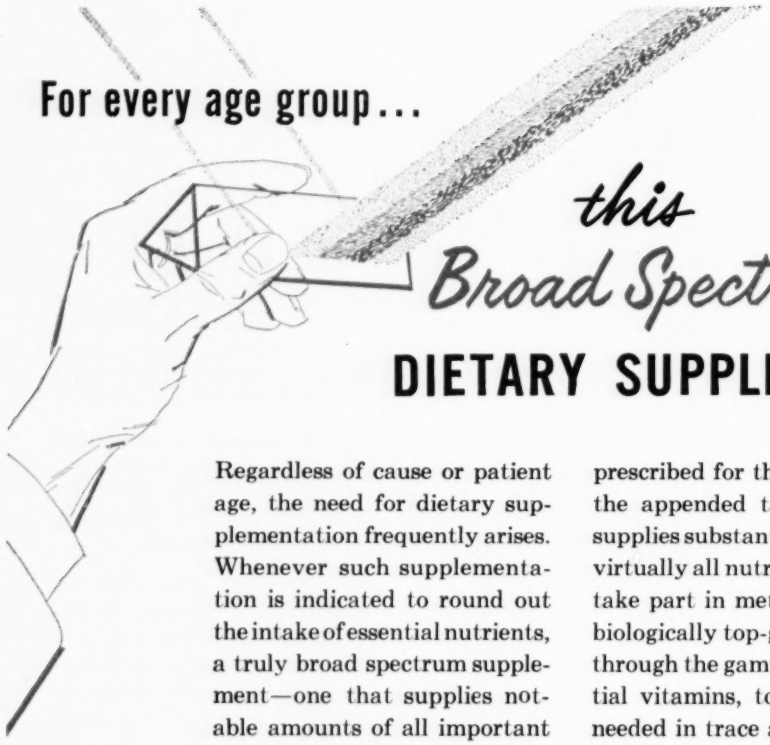

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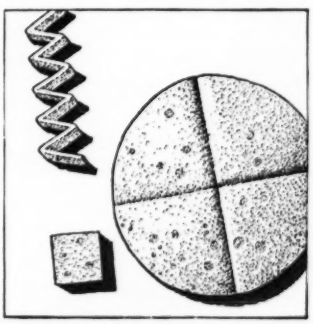

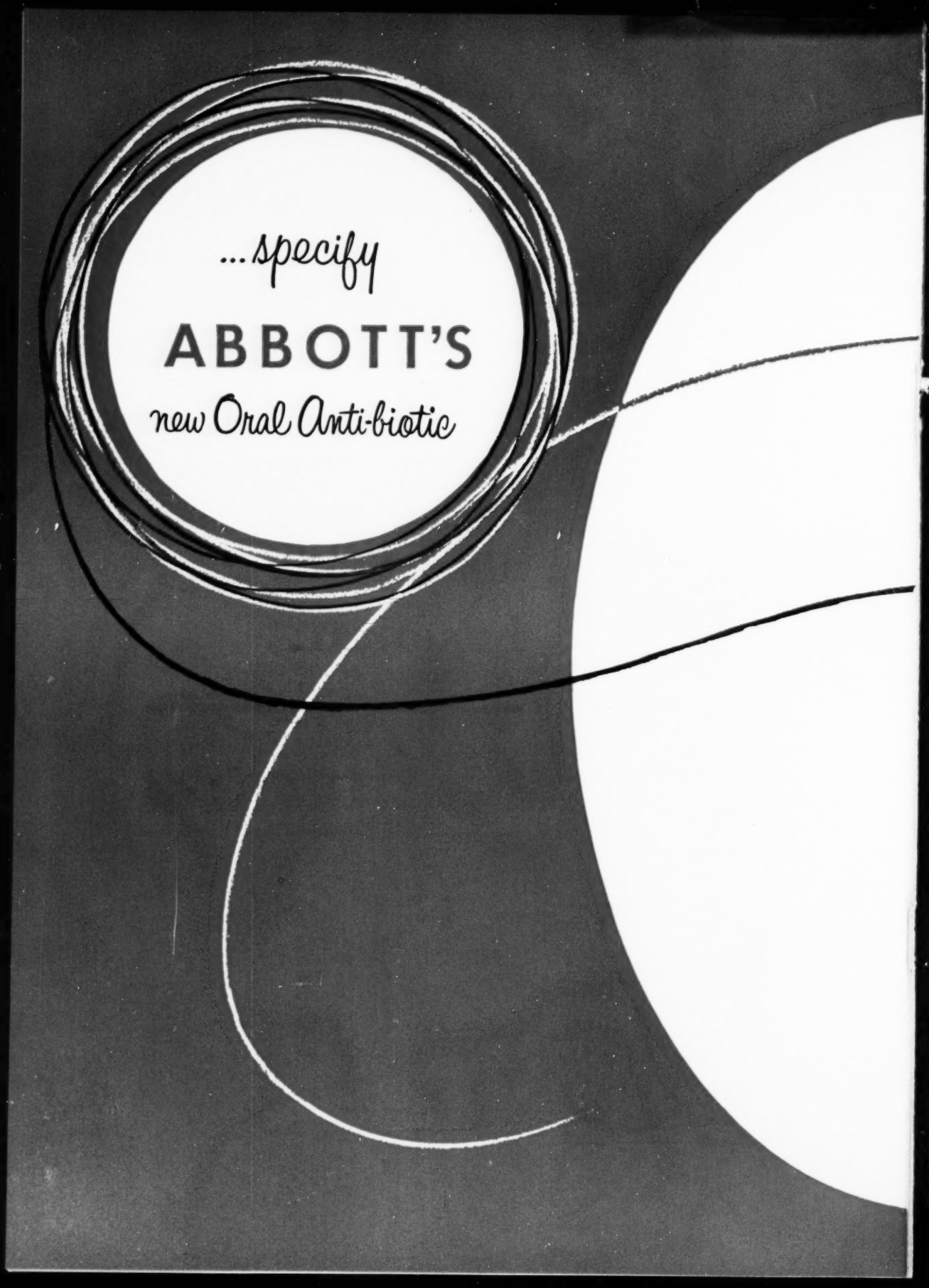
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1. McGuire et al. (1952), J. Antibiotics & Chemo., 2:281, June.
2. Heilman et al. (1952), Proc. Staff Meet. Mayo Clin., 27:385, July 16.
3. Haight and Finland (1952), New Eng. J. Med., 247:227, Aug. 14.

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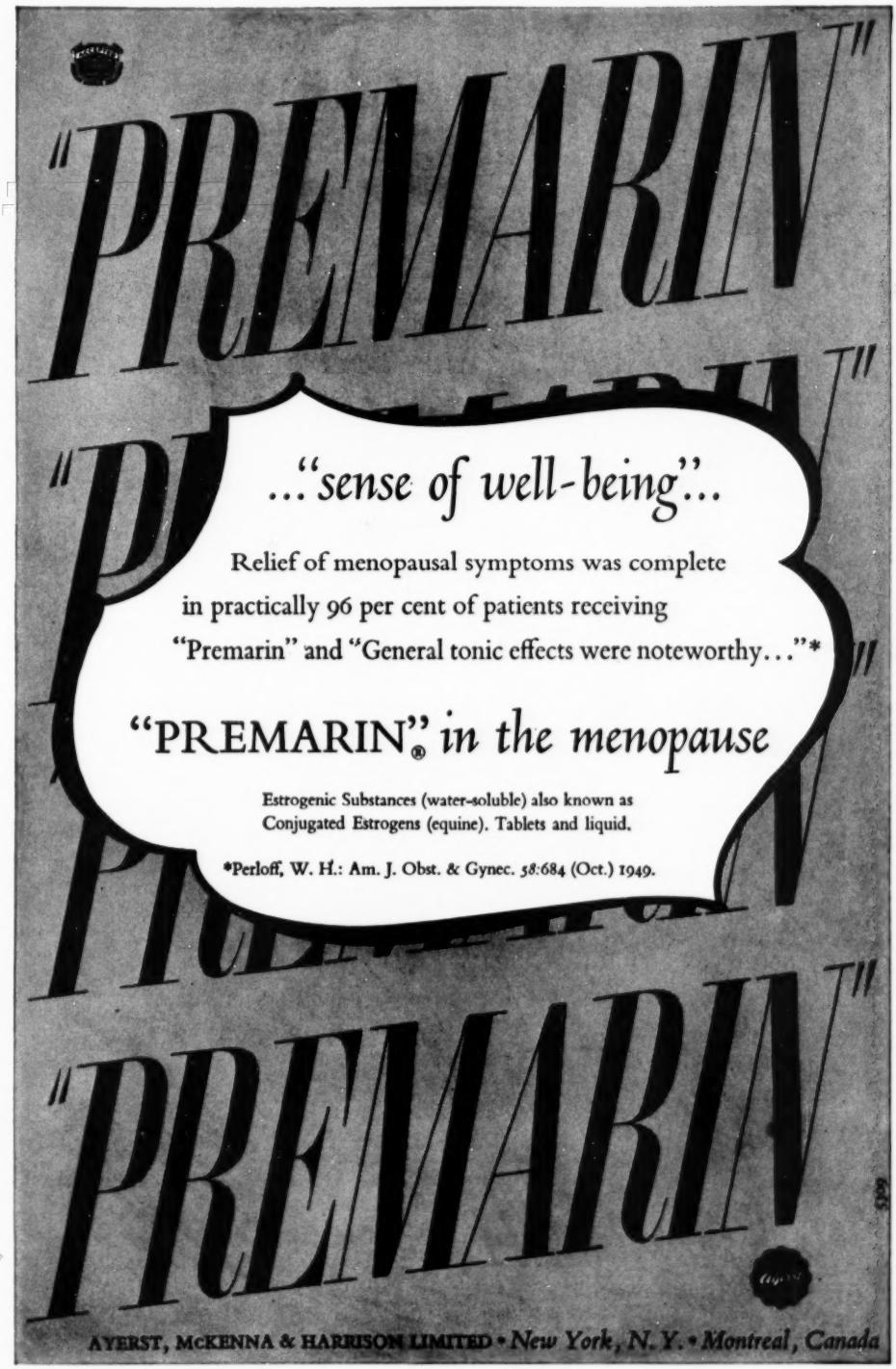
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*Perloff, W. H.: Am. J. Obst. & Gynec. 58:684 (Oct.) 1949.
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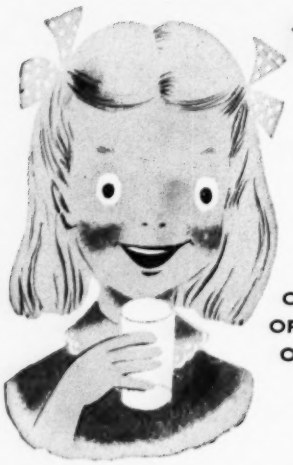

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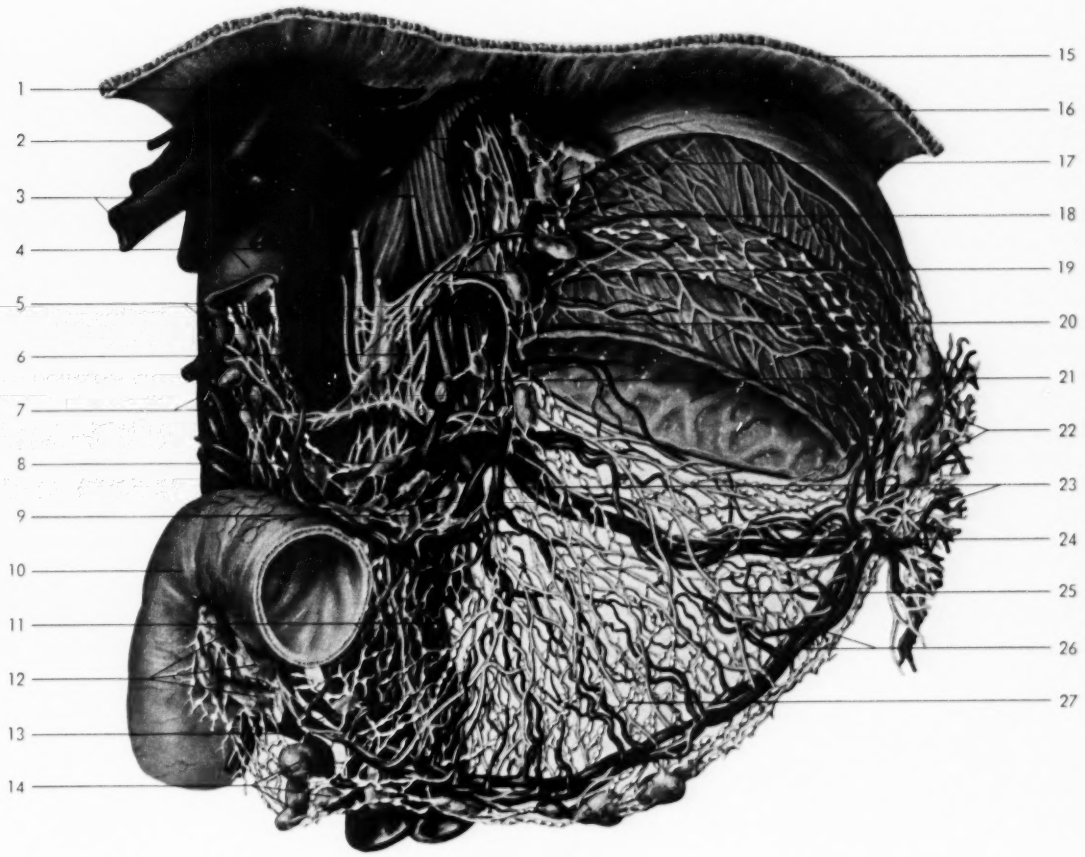

1 Middle and left hepatic veins

2 Right vagus nerve and esophagus

3 Right hepatic vein and crura of diaphragm

4 Inferior vena cava and greater splanchnic nerve

5 Portal vein and hepatic artery

6 Celiac plexus and celiac artery

7 Hepatic lymph node and hepatic rami of vagus nerve

8 Gastroduodenal artery and suprapyloric lymph nodes

9 Superior gastric lymph nodes

10 Duodenum

11 Superior mesenteric artery and vein

12 Subpyloric lymph nodes

13 Right gastroepiploic artery and vein

14 Inferior gastric lymph nodes

15 Diaphragm

16 Serosa

17 Paracardial lymph nodes

18 Left vagus nerve and longitudinal muscular layer

19 Abdominal aorta and circular muscular layer

20 Left gastric artery and oblique muscular layer

21 Celiac rami of vagus nerve and gastric mucosa

22 Splenic lymph nodes

23 Left gastric (coronary) vein and splenic rami of vagus nerve

24 Splenic artery and vein

25 Gastric rami of vagus nerve

26 Left gastroepiploic artery and vein

27 Gastric lymphatic plexus

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THE ELECTROCARDIOGRAPH ELECTROLYTES, AND TOXEMIA OF PREGNANCY

WILLARD F. PRESTON, M. D.,*
EDWARD M. BOHAN, M. D.,
and
J. RICHARD DURHAM, M. D.,
Wilmington, Del.

Myocardial damage occurring in pregnancy has interested observers for many years. In 1937, Gouley, McMillan, and Bellett[1] reported an idiopathic myocardial degeneration associated with pregnancy (and especially the puerperium) with autopsy findings revealing edema of the endocardium, round and epithelial cell infiltration, and foci of endocardial thrombosis. Pulmonary infarction was sometimes seen. The electrocardiograms showed inverted T waves, but were not diagnostic of a specific entity. In 1948, Wallace et al[2] reported two cases of toxemia with electrocardiographic changes suggesting atypical anterior infarction or strain. One case showed restitution in two weeks. He believed the electrocardiographic changes could be divided into those normally occurring in pregnancy due to change in heart position and hydremia, those due to toxemia per se, or those due to toxemia plus cardiac failure. Mendelson[3] reported two cases of isolated myocarditis in pregnancy; autopsy revealed both hearts to be enlarged, and he concluded that the etiology was obscure. In most cases of eclampsia, the heart was found to be more or less involved at autopsy and was perfectly normal in only eight out of one hundred and two autopsies analyzed by Polak.[4] According to Schmorl,[4] the changes usually consisted in degenerative processes in the myocardium. Disregarding cloudy swelling and fatty degeneration, which were frequently present, he found hemorrhage and necrosis in the myocardium in forty-three of seventy-three autopsies. In the neighborhood of the hemorrhages, the muscle fibres were disintegrated into hyaline layers usually with the nucleus gone and the transverse striations no longer recognizable.

In Way's[4] more recent studies of thirty-three cases, the heart was the site of hemorrhage in twelve patients; four of these showed focal necrosis of the myocardium with slight cellular reaction. There were two cases of myocarditis, one characterized by focal aggregation of polymorphonuclear leucocytes and the other a diffuse scattering of round cells.

Clinically, the frequency of pulmonary edema and cyanosis in severe eclampsia suggests heart failure; moreover, autopsy findings indicating cardiac damage is not uncommon. Stroganoff[4] was impressed with the important role played by heart failure in eclampsia and included digitalis in his regime of therapy. White stated that "there may be serious toxic myocardial dilitation with acute heart failure and pulmonary edema" in the acute hypertension associated with toxemia.

In 1946, Wallace et al[2] examined the electrocardiographic records of twelve patients suffering from toxemia and found significant changes in six with actual left ventricular failure in two of these. They felt that the alterations simulated those occasionally seen in acute nephritis and attributed them to local myocardial necrosis, infiltration and edema, rather than to myocardial infarction secondary to occlusion of the coronary artery.

Szelely and Snaith[4] made detailed observations of the heart in nineteen unselected cases of toxemia of pregnancy and found that seven cases showed significant clinical and/or electrocardiographic changes. Left ventricular failure was observed in three, and one of these also showed T wave changes similar to those seen in anterior myocardial infarction. Significant electrocardiographic changes were seen in five, these changes consisting of

* From the Department of Obstetrics and Gynecology, and of Medicine, St. Francis Hospital.

transient alterations of the T waves usually in both standard and chest leads.

On the other side of the question, Hamilton,[4] on the basis of a large experience with heart disease in pregnancy, has taken a very strong stand, stating "our present opinion is that uncomplicated pre-eclampsia or eclampsia causes neither heart failure nor significant acute myocarditis." Over the years, he has carried out a searching study of this problem and is still looking for a case where undoubted myocarditis of clinical importance appears during uncomplicated eclampsia or pre-eclampsia. In regard to edema of the lungs, he believes that this is due to causes other than cardiac weakness.

There is, therefore, a decided difference of opinion amongst cardiologists in regard to the role which cardiac alterations play in the pathologic physiology of this disease. This is most regrettable, since the question has to do with such important problems as the justification of digitalization in pre-eclampsia and eclampsia. Despite Hamilton's opinion, many obstetricians cannot help but be impressed by the abundant evidence documenting the contrary point of view, and for some years they have digitalized all eclamptic patients.

CASE REPORT

Mrs. C. D., twenty-two, referred to the St. Francis Hospital by Dr. A. R. Cruchley, a primipara in the sixth month, was admitted 2/18/52 having had one convulsion, plus edema of the ankles and hands with nausea and vomiting of one month's duration. Fatal heart sounds were heard at the rate of 140 per minute, and a labile hypertension was present, with readings over a nine day period varying from 122/80 to 170/112. The laboratory reported blood urea 23 mg.%, uric acid 5.5 mg.%, and total protein 5.6 gm. with albumin 4 and globulin 1.6. Repeated urinalyses showed 4 plus albuminuria with specific gravities running between 1.012 and 1.027. The patient seemed confused and responded poorly to questioning. Her muscle tonus suggested a pre-eclamptic state. This diagnosis was entertained and treatment with magnesium sulphate intramuscularly, low sodium diet, and intravenous fluids resulted in slight improvement. The patient was afebrile throughout this admission and was discharged 2/27/52. No electrocardiogram was taken.

On readmission 3/6/52, edema had increased, involving the ankles, legs, vulva, and buttocks. No dyspnea, cyanosis, or fever were present, although moist basal rales were detected. Heart size was normal by X-ray, but the costophrenic angles were partially obscured with possible effusion at the left base (see Fig. 1). No fetal heart sounds or movement could be detected, and blood pressure, particularly diastolic, was now persistently elevated.

Fig. 2 reveals serial electrocardiograms which could be interpreted as atypical anterior infarction or strain pattern. Daily urinalyses continued to show 4 plus albuminuria and some granular casts, but appropriate laboratory studies failed to reveal nitrogeneous retention, anemia, acidosis, or blood protein derangement as a factor in the STT changes. Opthalmological consultation (Dr. W. O. LaMotte, Jr.) showed no retinal changes.

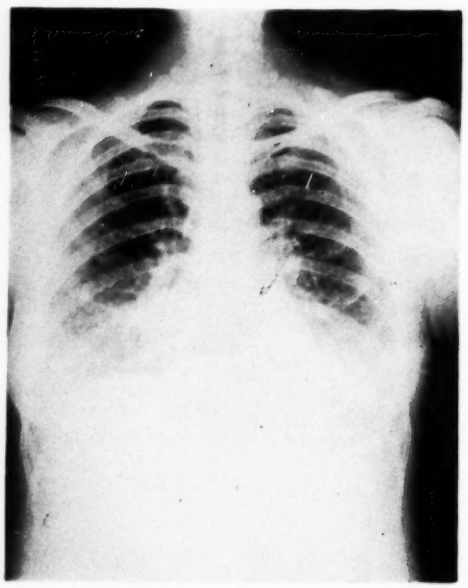

Fig. 1. Six foot plate showing normal heart size with possible effusion at left base.

On March 7th, the patient's condition had deteriorated considerably and it was felt that electrolyte studies should be determined. Since it is well known that the syndrome of eclampsia may be at times superimposed upon intrinsic renal disease, studies of serum elec-

trolytes were done. These revealed a low sodium of 129 mEq/L and a high potassium, 6.25 mEq/L, with a CO2 of 17 mEq/L and a chloride of 65 mEq/L. These determinations suggested a decrease in the osmotic pressure of the extracellular fluid, and the problem of restoring this arose. Since it is known that abnormalities of the extracellular fluid may

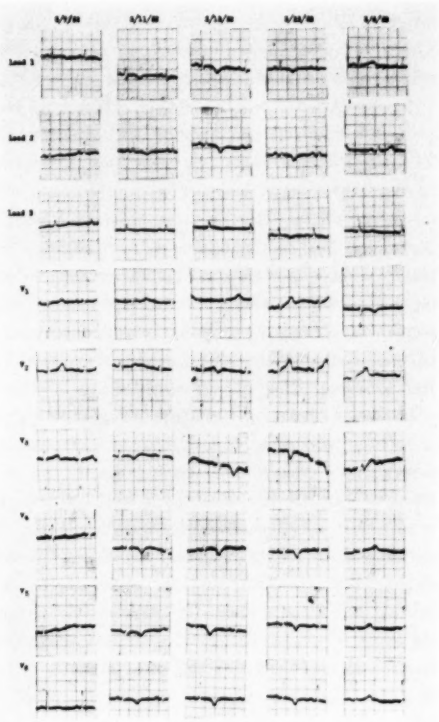

Fig. 2. Serial electrocardiograms. See text.

cause a further decrease in renal function in patients with advanced renal dysfunction, it was, therefore, decided to give this patient an isotonic infusion of sodium chloride lactate (750 cc. normal saline plus 250 cc. of 1/6 molar lactate). A more hypertonic solution was not chosen because of the fact that the content of 155 mEq/L of the isotonic solution was hypertonic at that time for the patient's serum. It was felt that this solution would tend to lower the serum potassium and mildly restore the structure of the extracellular fluid.

Immediately following the electrolyte infusion, the patient was symptomatically improved and able to eat her dinner. Several hours later, the patient was given 500 cc. of blood. Within 30 minutes of the end of the transfusion, the patient experienced acute respiratory distress and went into acute pulmonary edema. Following the blood that was administered, the patient became relatively oliguric, and passed only 1,200 cc. of urine in the next 32 hours. It is of note that several hours after the transfusion the patient spontaneously voided 50 cc. of blood tinged urine. Urinalysis the following day revealed a decrease in the albumin and an alkaline pH. It is felt that this represented a type of transfusion reaction and that the alkaline medium prevented further renal damage. In view of some confusion as to the cause of the acute congestive failure, further electrolyte therapy was not felt advisable.

Supportive therapy with digitalis, oxygen, and intramuscular magnesium sulphate was continued. No mercurial diuretics were used, and adequate urinary output returned. Fetal heart sounds were not elicited, and vaginal or rectal examination was impossible due to the marked edema of the parts. Because of the latter, the patient was taken to the delivery room, and under sterile precautions, the vulva and perineum were sacrificed with a knife, and glycerine and magnesium sulphate compresses applied to relieve the localized edema. These measures of dehydration proved so effective that within forty-eight hours, one of us (W.F.P.) delivered a macerated male fetus (thirty-two weeks gestation) spontaneously without laceration, under gas-oxygen-ether anesthesia. The placenta delivered complete.

Her postpartum course showed gradual return to normotension, disappearance of edema, and clearing of proteinuria. As will be observed in Fig. 2, however, her postpartum electrocardiograms of 3/18/52 and 3/22/52 continued abnormal, actually revealing greater T wave negativity than observed before. The patient was discharged clinically well on 3/22/52. Cardiovascular examination on 5/6/52 (see last electrocardiogram in Fig. 2) was entirely normal.

REVIEW OF THE LITERATURE

The reason for correction of this unusual state of dehydration plus edema in the same disease was based on the work of Turner, De-

Lozier and Whitacre,[5] who used isotonic normal saline to correct low sodium levels in eclampsia. If dehydration only is present, these writers do not routinely use normal saline, but 5% dextrose in distilled water at the rate of 60 drops per minute; neither do they give more than 1,000 cc. of the isotonic glucose solution in any six hour period although 4,000 cc. per 24 hours has been given.,

It must, therefore, be recognized that eclampsia and pre-eclampsia are fertile fields for electrolyte imbalance which may demand careful evaluation and therapy.

Electrolyte therapy has already taken its place in medicine and surgery, and there is some reason to believe that the obstetrical patient is prone to these metabolic changes, although there are other schools of thought, as referred to in later paragraphs.

Neubauer,[6] one of the pioneers in electrolyte therapy, says "salt restriction may be harmful at times, the tubules of the kidney losing salt in large amounts. This syndrome may occur in the presence of edema, and hypertonic sodium chloride of 3 to 5% may be necessary to enable the kidney to resume its normal function. The simplest urine for excretion is an iso-osmotic urine where little renal energy is required." He reports a case refractory to mercurial diuretics, responding to hypertonic sodium chloride plus mercurial diuretics with loss of nearly all of the edema.

In 1948, Fox[7] published a detailed description of serum and urine electrolyte changes in nephrosis, treated with the combined salts of sodium and potassium acetate. He was able to show that in the edematous phase of the disease there was a lowered serum sodium and that the urine was hypotonic for sodium.

Evidently our thinking must be revised in reference to edema. We must think in two dimensions; that is, in the intracellular and extracellular compartments, and furthermore, consider their relation to each other and to the intake and output of fluid and electrolytes.

In reviewing the literature on myocardial changes occurring in pregnancy, including clinical and pathological studies, one is impressed with the obscure nature of this malady. Since most of the reports were written before the days of electrolyte study, we believe this case with its sodium and potassium levels is worth recording. These electrolytes apparently played no part in the electrocardiographic changes, and, while calcium levels were not accomplished, the QT times do not suggest this to be a factor. The outstanding biochemical alteration in pre-eclampsia and eclampsia is water retention over and above that found in a normal pregnancy; indeed, the edema of the eclamptic syndrome may be regarded as an exaggeration of that often seen in normal pregnancy, and whatever causes the one is probably responsible also for the other. The accumulation of tissue fluids seen in eclampsia, in other words, represents simply a physiologic process gone awry. Water retention and sodium retention are so inseparably connected that it is not possible to state whether the edema seen in acute toxemia, as well as in normal pregnancy, represents a primary disorder of water or of sodium metabolism. The evidence that sodium chloride is retained in the toxemias is several fold. Rupp gave five grams of sodium chloride to normal pregnant women and to women suffering from the toxemias of pregnancy, and then investigated the resultant changes in the chlorides of the blood and urine, in order to deduce the fate of the salt. In normal non-pregnant individuals, the ingestion of this amount of sodium chloride was followed by a marked rise in the blood chlorides, together with a simultaneous increase in the urinary output of sodium chloride. In normal pregnant women, on the other hand, blood chlorides augmented only slightly after feeding while the urinary excretion of chlorides was delayed. In cases of toxemia with edema, this effect was still more marked, the ingestion of sodium chloride having no effect on either the blood or urinary chlorides—all of the ingested salt being held by the tissues. Finally, Rupp[4] analyzed the edema fluid of these patients, and showed that in cases of toxemia with edema, the ingestion of salt caused a characteristic increase in the sodium chloride content of this fluid.

The observations of Hardy and VanWyck,[4] made some years ago, are also pertinent to this problem. The authors gave several patients, who were suffering from various

grades of toxemia of pregnancy, intravenous injections of 300 cc. of six per cent sodium chloride solution in the belief that this might be a valuable therapeutic measure. The results were well-night disastrous. One patient, who had received two such injections within twenty-four hours developed a fulminating pre-eclampsia with dramatic suddenness, and worse still, went shortly into actual eclampsia and had three typical convulsions. Meanwhile, the blood pressure had risen from 128/100 to 200/140 and the albuminuria had more than doubled in intensity. The patient recovered, but the authors wrote that they had no wish to continue their observations in this direction. They believed firmly that a high intake of salt ingested at the right moment in a developing toxemia will produce albuminuria, increase the blood pressure, and even cause convulsions in a short period of time. Renal function studies indicate a decreased ability to concentrate sodium chloride during pregnancy, an impairment which is intensified in toxemic patients.

Concerning the effect of magnesium, Stanburg and Farah[8] have demonstrated that this ion can decrease the heart rate by direct action on the SA node and may abolish extrasystoles or abnormal rhythms in overdosage of digitalis preparations. In dogs, the cardiac output was decreased, but coronary flow and stroke volume were increased. The effects obtained paralleled serum magnesium levels and calcium ions reversed its effects. No electrocardiographic changes were reported by these authors.

In fulminating pre-eclampsia (as well as in eclampsia) magnesium sulphate intramuscularly or intravenously is regarded as a most valuable agent and this is attested by the eperience of numerous clinics over many years. The efficacy of magnesium sulphate as a central nervous system depressant and as a vasodilator is well known and is substantiated by a large amount of experimental and clinical evidence. In 1925, Blackfan and Hamilton[8] observed that intravenous injections of magnesium sulphate produced a favorable influence by lowering the blood pressure and increasing the flow of urine in children suffering from hemorrhage nephritis. These observations have been confirmed by numerous workers and, more recently, Haury has shown that intravenous injections of magnesium sulphate produced a marked dilitation of the splanchnic organs including the kidneys. Since both sedative and vasodilator effects of magnesium sulphate are urgently to be desired in the treatment of pre-eclampsia and eclampsia, it is not surprising that this drug has been widely employed in the management of these conditions over the past twenty years.

It is important to recall that toxic signs and symptoms do not appear until the serum magnesium reaches 10 to 12 mgs. per 100 cc. At, or near this level, the knee jerks disappear. Between 12 and 15 mgm. per 100 cc., respirations are likely to cease, and when the serum magnesium concentration reaches 15 mgms. per 100 cc., cardiac action may stop and death ensue. Eastman has employed this drug in over four hundred cases with rather large total dosages, and believes that it is possible to administer magnesium sulphate intramuscularly in several times the dosage usually employed without approaching hazardous levels of serum magnesium. On the basis of his experience, which includes more than 300 serum magnesium determinations done at various times after the administration of the drug, he feels, in the first place, that it is entirely safe to administer 10 gms. intramuscularly as an initial dose, followed by 5 gms. every six hours. He has continued this for as long as two or three days with total dosages reaching 60 to 110 gms. and never has observed a serum magnesium level of above 7 mgms. per 100 cc. Usually, the range obtained by such dosage is between 4 and 6 mgms. per 100 cc. In the second place, it is his impression that such levels of magnesium contributed substantially to the lowering of the blood pressure and prevention of convulsions.

In this case report, no clinical signs of magnesium intoxication were encountered, and, although no serum levels were obtained, the electrocardiograms continued to show abnormalities both before and after the use of magnesium. Smith, et al,[9] reporting on electrocardiographic changes following intravenous magnesium in dogs, found AV and intraventricular block patterns produced by

high dosages of this ion, but with no T wave changes of any moment. Hence, it appears that this electrolyte had no bearing on the changes in the tracings.

It is our belief that absorption of toxic products from the fetus may in some manner damage the myocardial cells and effect repolarization, which, in this case, proved reversible. It is hoped that further study of similar cases will lead to a better understanding of this abnormality.

BIBLIOGRAPHY

1. Gouley, R. A., McMillan, T. M., Bellet, R.: Idiopathic myocardial degeneration associated with pregnancy and especially the puerpium. Am. J. Med. Sci. 194:2, 1937.
2. Wallace, L., Katz, L. N., Langendorf, R., Buxbaum, H.: Electrocardiograms in toxemia of pregnancy, Arch. Int. Med. 77:405-419, April, 1946.
3. Mendelson, L. C.: Acute isolated myocarditis in pregnancy, Am. J. Obst. and Gyn. 61:1341, June, 1951.
4. Cited in Williams: Textbook of Obstetrics (Eastman-3rd edition). New York: Appleton-Century Company, 1952.
5. Turner, H. L., DeLozier, O., Whitacre, I. E.: Hydration treatment of eclampsia, J. A. M. A., 148:12, March 22, 1953.
6. Neubauer, R. A.: Some changing concepts of sodium metabolism in cardiac and renal diseases, Delaware State Medical Journal 24: 4:98, April, 1952.
7. Fox, C. L., Jr., McCune, D. J.: Electrolyte changes in nephrosis, Am. J. Med. 216:1 (1948).
8. Cited in Year Book of Obstetrics and Gynecology. Chicago: Year Book Publishers, Incorporated, 1953.
9. Smith, P. K., Winkler, A. W., Hoff, H. C.: Electrocardiographic changes and concentration of magnesium in serum following intravenous injection of magnesium salts, Am. J. Phys. 126:720, 1939.

FURTHER OBSERVATIONS ON THE USE OF THE POST-PRANDIAL BLOOD SUGAR IN THE TREATMENT OF DIABETES MELLITUS

Apparent Cure of Three Obese Diabetics

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ADVANTAGES OF POST-PRANDIAL SUGAR ESTIMATION

Since the original publication of Sindoni[1] on post-prandial sugar in diabetes mellitus and its comparison to fasting sugar, the use of the post-prandial sugar has become more prevalent in the diagnosis and treatment of the disease. In 1939 the relationship between blood sugar and urine sugar was clearly described by him and it was concluded that the use of the blood sugar as a guide[2] in the treatment of this disease was far superior to the urinanalysis. At this time he said "in view of the high incidence of disturbed carbohydrate metabolism frequently escaping detection by examination of the urine for sugar and the serious consequences resulting therefrom, blood sugar estimations, fasting and after meals, should be indispensable in early and latent recognition and treatment of diabetes mellitus." When one realizes that about 11% of diagnoses are missed by taking fasting sugars, there seems to be a need for greater attention on our part to the use of this method of diagnosis and treatment. The attention should be directed not only to the physicians of America but also to the doctors of the world. When one gets 'feasting' sugar conscious, amazing revelations come to our minds. One of these is the normal post-prandial sugar. What is its value? This is found in the writings of Sindoni,[1,2] and also in a later paper by Ackerman and Bohan[3] which only served to follow his teaching and verify the work originally done by him. The blood sugar after meals does not exceed the fasting level of 120 mg. in normal persons. Recently a lady with a diabetic family history had a fasting blood sugar of 85 mg. per 100 cc. of blood but a post prandial sugar of 140 mg. This reading is abnormal. A glucose tolerance test done under standard conditions (previous three day diet of at least 300 gm. of carbohydrate) was definitely abnormal. This is an example of similar cases observed at the Philadelphia General Hospital, St. Joseph's Hospital, St. Agnes Hospital in Philadelphia and St. Francis Hospital in Wilmington by Sindoni, Gerber, Bove[4] and the authors.

In the treatment of diabetes mellitus with the use of the post-prandial sugar an attempt is made to keep the sugar level between 170 mg. to 220 mg. per 100 cc. of blood in the insulin treated patient. This is the compatible level described by Sindoni,[1] the level at which the patient is comfortable and in good general health without diabetic symptoms. In a scientific paper to be published soon, he reports 53% of his patients discontinuing insulin with this method of treatment.

THE OBESE DIABETIC

During the treatment of three obese diabetics with diet and insulin and employing the post-prandial sugar as a guide, it was found as the weight was reduced, the insulin was discontinued, and the post-prandial sugar returned to normal levels with apparent cure

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of the diabetes. This so-called cure has been reported before, but not using the post-prandial sugar as a guide in treatment.

The relationship of obesity to diabetes is well known and is often interpreted as one of the causes of the disease. Varying estimates place the percentage of diabetes occurring in the obese person between 70 and 80%. The disease is eight times commoner than in those of average weight. Ten thousand children are less likely to have diabetes than ten middle-aged obese persons. Stone[5] reports twenty-five per cent of the population of the United States to be overweight. The frequent association of gall bladder disease and diabetes may partly be due to the obesity as a common denominator although the former is nine times more common when the patient is a diabetic.[6] Favorite words of Elliott Joslin[7] in discussing obesity as an etiological factor in diabetes are "fat man's folly" and "open door to diabetes."

The history of past overweight may be of great importance in classifying the type of diabetic one is dealing with. Loss of weight may have occurred with an already existent and uncontrolled diabetes. In a recent analysis of the weight of persons developing diabetes after forty, it was found that 60% had previously been markedly overweight and an additional 25% had been moderately overweight. Only 15% did not give a history of obesity preceding the diabetes. When detection of hyperglycemia has been made an effort should be made to classify the type of the diabetes. Ucko[8] makes five classifications:

a. Juvenile or brittle type.
b. Hepatic type: hyperglycemia of liver origin.
c. Climacteric: diabetes occurring at this age.
d. Bronze diabetes or hemochromatosis: an iron metabolic disorder.
e. Endocrine type: hyperglycemia seen in hyperthyroidism, hyperpituitarism, and hyperadrenalism.

To this outline may be added two more human types of the disease: the obese type, since it does not fall under the endocrine classification except in certain cases, and those cases due to direct invasion of the pancreas by inflammation or tumor.

Another classification,[9] physiologic in scope, is sometimes made according to the insulin content in the human body, which deficiency, may be absolute or relative:

a. Hereditary deficiency of beta cells of the pancreas.
b. Destruction of the beta cells by (1) pharmacologic agents, e.g. alloxan. (2) disease as chronic pancreatitis or tumor of the organ.
c. Exhaustion of beta cell activity because of an excessive demand for insulin, e.g. obese type of diabetes.
d. Deficiency of insulin precursors as glutathione (a suggestion was made of amino acid deficiency as a possible cause of insulin deficiency in a paper published by one of the authors[10] in 1947.).
e. Extra-pancreatic causes may be due to antagonism to insulin from its mates in the glandular system, e.g. anterior pituitary, adrenal cortex or thyroid gland, or to peripheral inactivation of insulin with failure of end organ response.

Evidence of correlation between maternal obesity of a progressive type, the birth of unusually large babies and ensuing maternal diabetes has been demonstrated by Gilbert.[11] He suggests that excessive secretion of the growth factor during pregnancy may account for these associated phenomena. Growth hormone has been shown to be the most important anterior pituitary factor outside ACTH. Its natural action is to inhibit the peripheral utilization of sugar, thereby antagonizing the action of insulin.

An interesting study was made in Scotland on 413,110 Scottish recruits during the last World War. Munro, Eaton and Glen[12] found no relationship between the presence of a positive family history and the severity of the disease. The frequency of diabetes with a positive family history in obese and hypertensive patients did not differ from that of the group of diabetics as a whole. The hereditary factor does not seem to be concerned with obesity but more with the diabetic status of the ancestry. If neither parent has been identified as a carrier or a potential diabetic, the chance of the child developing diabetes is theoretically zero.

Since the greatest single cause of obesity is overeating, it is readily seen why the in-

cidence of diabetes is reduced in rationing periods as in France during World War I. On the other hand, because the children of most families tend to follow the eating habits of their parents, eating more by rhyme than reason, the tendency of obesity is to be familial. Obesity may also be induced by the endocrine pattern set into many families. Hypothalamic dysfunction may greatly dispose to obesity. Overactivity of the anterior lobe of the pituitary or of the adrenal cortex are known to be associated with obesity. All of these observations suggest that a reason for the obesity should be searched for when diabetes and obesity are found to be associated. For example, it is easy to miss an adrenal tumor which can be diagnosed more easily today than a few years ago.

The obese diabetic usually follows a mild even course as compared to the juvenile or brittle type of diabetes. It is Short's[13] opinion that the diabetic state in obesity is due to pancreatic exhaustion. John[14] believes that this destruction of the islets of Langerhans is reversible in the early stages. Long[15] says that the obesity brought on by overeating deposits fatty acids in the depot areas of the body. These fatty acids require insulin over and above that needed to maintain weight. He believes that the abnormal strain on the pancreas in the obese diabetic virtually amounts to a partial pancreatectomy. Lawrence[16] of England has a different theory as to the high incidence of diabetes in the obese middle-aged man or woman. He suggests that the hyperglycemia and glycosuria are a reflection of the deficient capacity of the overloaded fat cells to form more fat from carbohydrate. Thirty years ago Allen and his coworkers[17] found that a partial pancreatectomy in dogs which had no apparent effect when they were eating in the usual way was followed by diabetes when the dogs were fattened by overfeeding.

In the detection of diabetes the chances are much better of finding the diabetic in the obese, and in those hereditarily prone to the disease. A study made of 25 known or suspected cases of diabetes by Ackerman at the Savannah River Plant of the DuPont Company disclosed 12 with abnormal blood sugars at the ½ hour to three hour post-cibal level. Of the 12, 9 or 75% were overweight.

APPARENT CURE OF DIABETES

Diabetes in some people is subject to wide variations in severity. It often becomes milder in associated kidney disease, in jaundice or following a cerebral accident. These variations are not in the same category as apparent cure. The latter means that to all intents and purposes the diabetic state is indetectable by present known methods of diagnosis. Glassberg[18] reports apparent cure in a case of diabetes associated with cirrhosis of the liver. In his case there seemed to be some evidence of a relationship between body weight and the improvement in the glucose tolerance curve, the curve improving as the weight dropped. The person with apparent cure retains the potentiality to develop the diabetic state again.

Since three cases of this type were seen by the authors within the past two years, it was thought worthwhile to report them. All were followed with post-prandial blood sugars. No glucose tolerance tests were done because they seemed to be frank diabetics to start with and after their post-prandial blood sugars were found to be normal over a period of months glucose tolerance was not done because of the danger of upsetting the delicate carbohydrate metabolism.

CASE NO. 1

A male diabetic age 55 years. Height 5' 10''. Weight 228 lbs. He had no knowldege of the disease until he was brought into St. Francis Hospital in diabetic acidosis. He had been drinking large quantities of fluid for the past three weeks. His family history was negative for diabetes. Blood sugar on admission was 750 mg. . . CO2 was not taken until the next morning and was 40 volumes per cent. With intensive therapy for acidosis the patient made a gradual recovery over a period of approximately three weeks. The following

table shows the trend of the blood sugars and the gradual reduction of weight. With 35 lb. weight loss the post-prandial blood sugar became normal and has remained at that level since that time.

Insulin and admitted to St. Francis Hospital. Marked visual disturbance resulted from the insulin which was discontinued before the patient left the hospital. After becoming an office patient weight reduction was steady and

TABLE 1

HOSPITAL ADMISSION

Date	*Blood Sugar mg. per 100 cc*	*Time*	*Symptoms*	*Weight (lbs.)*	*Daily Insulin Units*	*Other Notes*
1951						
8-27	750 mg.	3 PM	Coma	228	320 plain	Blood urea 45 mg.
8-27	500 mg.	11 PM	Semi-coma	.....	....	....
8-28	400 mg.	1 AM	Stupor	.....	227 plain	CO2 40 vol. %
8-28	72 mg.	9 AM	Thirsty	.....	....	Urea 72 mg.
8-28	150 mg.	1 PM	Aroused	.....	....	....
8-29	450 mg.	8 AM	Disoriented	.....	238 plain	Plasma acetone negative
8-30	530 mg.	8 AM	Disoriented	.....	140 plain	
8-30	320 mg.	1 PM	Improving	.....	....	Urinanalysis Alb. + + Sug. + + micro. neg.
8-31	265 mg.	8 AM	Improving	.....	130 plain	Urea 19 mg.
8-31	220 mg.	3 PM	Improving	.....	....	....
9- 1	147 mg.	8 AM	Improving	.....	100 plain	X-Ray chest normal
9- 1	215 mg.	3 PM	Improving	.....	....	....
9- 2	320 mg.	post-prandial	Improving	.....	85 Globin	Diet full diabetic
9- 3	194 mg.	" "	None	.....	90 Globin	
9- 4	276 mg.	" "	"	.....	70 Globin	
9- 5	194 mg.	" "	"	.....	65 Globin	Skull X-Ray negative
9- 6	276 mg.	" "	"	.....	70 Globin	Pituitary negative
9- 7	194 mg.	" "	"	.....	60 Globin	Diet: 100 P 60 F 150 CH
9-10	164 mg.	" "	"	.....	60 Globin	
9-13	187 mg.	" "			40 Globin	
			OFFICE FOLLOW-UP			
9-20	130 mg.	p. p. 2 hrs.		210	25 Globin	
9-24	133 mg.	p. p. 2 hrs.		206	20 Globin	Diet same
9-30	84 mg.	p. p. " "		193	12 Globin	
10 5	86 mg.	p. p. " "		192	8 Globin	
10-11	90 mg.	p. p. " "		191	No insulin	
10-17	84 mg.	p. p. " "		188	" "	
10-31	93 mg.	p. p. " "		187	" "	
1-10 52	106 mg.	p. p. " "		183	" "	
3- 2-52	80 mg.	p. p. " "		186	" "	
10- 9-52	112 mg.	p. p. " "		186	" "	
1-20-53	96 mg.	p. p. " "		188	" "	

CASE NO. 2

A male patient, age 52. Height 5' 6½". Weight 180 lbs. Family history revealed one sister with diabetes. A blood sugar was taken because of extreme thirst, loss in weight of 15 lbs., in two months and itch. Fasting sugar 550 mg. Patient was given ten units of Globin

he was maintained on this reducing regime. Following a weight loss totalling 24 lbs., the blood sugar remained at normal level postcibally. The following table shows the trend of his successful loss in weight and the apparent cure of the diabetes.

TABLE 2

HOSPITAL ADMISSION

Date	*2 hr P. C. Blood Sugar mg. per 100 cc*	*Symptoms*	*Weight (lbs.)*	*Daily Insulin Units*	*Other Notes*
1950					
9-18	550 mg.	Very thirsty, itch, loss in wt.	180	10 Globin	Diet P 100 F 60
9-19	155 "	Slight Thirst		15 "	CH 150
9-20	201 "	" "		10	
9-21	160 "	Improved		10 "	Marked visu-
9-25	110 "	No symptoms		10 "	al disturb-
9-27	125 "	" "		8 "	ance
9-28	120 "	" "		6 "	Myopia
10- 2	137 "	" "		0	Vision better off insulin
10- 3	132 "	" "	176	0 "	Discharged
		OFFICE FOLLOW-UP			
10-10	128 mg.		173		Diet
10-17	130 mg.	No	173	No	as
10-24	108 "		171½		above
10-31	126 "	Symptoms	170	Insulin	
11- 7	83 "		166		
11-14	132 "		164		
11-21	115 "		162		
11-28	102 "		166		
12-31	98 "		163		
1951					
1- 4	113 "		161		
2- 5	115 "		160		
3- 8	108 "		158		
5- 2	112 "		157½		
8- 6	86 "		156		
12- 2	110 "		157		Diet
1952					P 110
2- 6	104 "		155		F 70
1953					CH 180
1-11	98 "		156		CH 20 H. S.

CASE NO. 3

A male patient, age 42. Height 5' 6". Weight 192 lbs. whose mother and brother have diabetes was admitted to St. Francis Hospital for surgical repair of an inguinal hernia. A diagnosis of diabetes mellitus was made, after the history revealed thirst, weakness and 15 lbs. of weight loss. The diabetes was brought under control and the patient returned for the operation at a later date. Insulin was discontinued and the patient, apparently cured, returned to normal blood sugar with absence of symptoms of diabetes. Table 3 shows the course of the disease in this patient, with the same result as in Tables 1 and 2.

COMMENT

Three obese diabeties who had initial blood sugars between 360 mg. and 750 mg. were reduced in weight until the blood sugars, 2 hours posteibal, were in the normal range (80-120 mg.). It was found that so long as the weight did not increase the sugars remained normal. No symptoms or signs of diabetes mellitus remained. After stabilization with unmodified insulin, globin insulin was used as the long acting insulin in maintaining control.

The use of the post-prandial sugar in treatment was found to be a satisfactory guide. No urinalyses were used to check the course of the disease.[2] After discharge of the patients from

TABLE 3

HOSPITAL ADMISSION

Date	*2 Hr. P.C. Blood Sugar mg. per 100 cc*	*Symptoms*	*Weight (lbs.)*	*Daily Insulin Units*	*Other Notes*
1950					
11- 5	360 mg.	Thirst-polyuria	192	10 units Globin	Original weight
11- 8	302 mg.	" "		10 " "	average
11-10	264 "	" "		10 " "	200 lbs.
11-13	322 "	No symptoms		20 " "	Urine acetone +
11-15	142 "	" "			Diacetic Acid Acid + + + +
11-16	266 "	Day of operation			
11-18	205 "				Acetone + Diacetic Acid + +
11-21	260 "	No symptoms		10 units Globin	Plasma acetone negative 11/21
11-22			187½	10 units Globin Insulin on discharge	Discharged 11/22 Diet 120 P 70 F 220 CH
		OFFICE FOLLOW-UP			
12- 3	208 mg.	No Symptoms	183	10 units Globin	Diet 90 P
12-15	142 "		181	8 " "	70 F
12-24	102 "		179	5 " "	160 CH
1951					
1- 2	96 "		177	No insulin	
1-30	–92 "		176½	" "	
2-27	–82 "		173	" "	
4- 3	–100 "		172	" "	
6- 8	–107 "		174	" "	
9- 7	–93 "		176	No insulin	
1952					
1-22	–85 "		178	" "	Diet
2-15	–84 "		178½	" "	100 P
3- 7	–82 "		179	" "	80 F
6- 9	–90 "		179	" "	180 CH
12-31	–102 "		177¾	No insulin	

the hospital each case was also followed by observation of the subjective and objective symptoms and signs. One patient lost 13 lbs. before the blood sugar became normal, the second case 12 lbs. and the third one 35 lbs., although 10 lbs. of the weight loss in the latter was undoubtedly due to the severity of the diabetes. Apparent cure has been reported before in the obese type of diabetes. Nutrition Reviews[19] report reduction of weight in 31 obese diabetics taking insulin. Six returned to normal glucose tolerance and 15 were able to discontinue insulin entirely.

CONCLUSIONS

1. Further observations on the use of the post-prandial sugar lead us to believe that it is superior to the fasting sugar as a guide in the treatment of diabetes mellitus. Its value in diagnosis is unquestionably superior—11% more diagnoses being made by its use. Since the normal post-prandial sugar is not over the fasting level[1,3,20] the attainment of this level

in the three cases reported gives us information that could not be obtained by employing the fasting blood sugar alone. The return to normal post-prandial levels gave us an excellent index of the patients' metabolizing power, after a meal was taken. Sindoni[1] has emphasized this latter point, as one of the important reasons for treating diabetics with the postcibal sugar as a guide. He also states that the fasting sugar 8-12 hours after a meal gives us absolutely no information of this character, being too far removed from the previous meal.

2. The frequency of obesity occurring with diabetes is often underestimated. Nine out of ten diabetics, whose diabetes developed after the age of twenty, have had obesity before the onset of the disease.[21] When the obese diabetic patient understands the danger of his overweight state in terms of mortality and morbidity, he should have a good incentive to reduce. If he is made to realize the frequency of the vascular complications of the diabetic state, and how these complications can be reduced by getting himself in the best possible physical condition, then a great service will be done. The probability of reducing the blood sugar to normal by the weight reduction is also a good selling point and may make the patient as happy as the three cases reported in this paper. If difficulty is encountered in weight reduction, there is no contraindication to the use of the anorexigenic drugs as an aid to caloric restriction.[19]

3. Periodic preventive medical care is necessary in cases of this type, but no more so than with all types of diabetes mellitus. The disease in its insidious but harmful course can be subdued, not only by reduction of weight, but also by the application of the general hygienic measures which have come to us through the years of trial and error, diet therapy and research. Insulin and diet are not the sole answers to the diabetic problem. The teamwork of the physician, dietitian, dentist, podiatrist and medical specialist, used preferably in the preventive capacity is the best solution we have today to the problems generated by this mysterious disease.

REFERENCES

1. Sindoni, A. M., Jr.: Fasting Blood Sugar versus Post-Prandial Sugar as Observed in Normal Individuals, Medical (Non-Diabetic) Patients, and Patients with Diabetes, Amer. J. Dig. Dis., 13: 178-192 (June, 1946).
2. Sindoni, A. M., Jr.: Blood Sugar versus Urine Sugar, JAMA, 112:2503-2508 (June 17) 1939, and 112: 2595-2600 (June, 24) 1939.
3. Ackerman, J. L., Bohan, E. M.: The Post-Prandial Blood Sugar, A Probable Normal, Del. St. Med. J., 23:122, (May) 1951.
4. Sindoni, A. M., Jr., Gerber, P., Bove, F.: Personal communication.
5. Stone, C. T., Chicago M. Soc., Bull, 53:755 (March 31) 1951.
6. Bohan, E. M.: Diabetes Mellitus and Gall Bladder Disease. Del. St. Med. J., 19:41 (March) 1947.
7. Joslin, E. P. et al; Treatment of Diabetes Mellitus, 8th Edition. Philadelphia: Lea & Febiger, 1940.
8. Ucko, H.: Endocrine Diagnosis, Staples Press, 1:361, 1951.
9. The Management of Patient with Diabetes Mellitus, Seminar, (Sharpe & Dohme), 12:4, 5, (Nov.) 1950.
10. Bohan, E. M.: Diabetes Mellitus and Cirrhosis of the Liver, Del. St. Med. J. 19:212, (Nov.), 1947.
11. Gilbert, J. A. L.: The Association of Maternal Obesity, Large Babies & Diabetes, British Medical Journal, 1:702-04, (April 23) 1949.
12. Munro, H. N., Eaton, J. C., Glen, A.: Survey of Scottish Diabetic Clinic—A Study of the Etiology of Diabetes Mellitus, J. Clin. Endocrin: 9:48-78 (Jan.) 1949.
13. Short, J. J.: Overall Management of Diabetes Mellitus, Post. Grad. Course, Endocrinol., 279:84 (Feb. 23-28) (1948).
14. John, H. J.: Diabetes, Pp. 60. St. Louis: C. V. Mosby Company, 1946.
15. Long, C. N. H.: Endocrine Control of the Blood Sugar. Diabetes, 1:6 (Jan., Feb.) 1952.
16. Lawrence, R. D.: Lipoplethoric Diabetes, Brit. Med. J. 1:373, 1951.
17. Allen, F. M., Stillman, E., and Fritz, R.: Total Dietary Regulation in the Treatment of Diabetes, N. Y. Rockefeller Institute for Scientific Research.
18. Glassberg, B. Y.: Apparent Cure of Diabetes, J. Clin. Endocrin, 6:369, 1946.
19. Nutrition Reviews: 9:199 (Jan.) 1951.
20. Orent-Keiles, E., Hallman, L. F.: Breakfast Meal in Relation to Blood Sugar Values, Circular No. 827: Dept. of Agriculture, Washington (Dec.) 1949.
21. Joslin, E. P.: Diabetic Manual, 243, Philadelphia: Lea and Febiger, 1953.

SURGICAL CONSIDERATIONS OF THROMBOEMBOLIC PHENOMENA*

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In discussing the surgical aspects of thromboembolic phenomena those acute conditions that are secondary to thrombosis and embolism in the venous and arterial systems will be considered. Chronic diseases of the arterial system, such as thrombosis secondary to arteriosclerosis, thromboangiitis obliterans, and periarteritis nodosa or surgical considerations of Raynaud's disease, and vascular lesions secondary to cervical rib will not be discussed. Regarding the venous system, we will be concerned with venous thrombosis of various types, and in the arterial tree with the acute embolus.

The terms "thrombophlebitis", "phlebitis" and "venous thrombosis" have been used interchangeably for several years. In the literature at the present time, two distinct types of venous thrombosis are recognized: (1) thrombophlebitis; (2) phlebothrombosis.

In order to have a working basis for discussion of various types of thrombotic disease

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occurring in the venous system, I would like to review briefly a clinical classification of thrombophlebitis based on etiology.

CLINICAL CLASSIFICATION OF THROMBOPHLEBITIS BASED ON ETIOLOGY

I. **Local thrombophlebitis**—due to direct injury to vein of the wall or pre-existing known disease of the wall in which thrombosis occurs
   (a) **Chemical thrombophlebitis** — sodium morrhuate, sodium ricinoleate, quinine and urethane, hypertonic solutions such as glucose and saline
   (b) **Mechanical thrombophlebitis**—occurs at site of contusions, lacerations, fractures, from strain or effort ("thrombophlebite axillaire par effort")
   (c) **Local inflammatory thrombophlebitis**—present in smaller veins as part of inflammatory non-suppurative lesions (Tb, lues, granulomas, etc.)
   (d) **Suppurative thrombophlebitis**—result of involvement of the wall of the vein by a severe suppurative disease caused by such organisms as streptococci, staphylococci, and pneumococci. Thrombus is highly infective and contains many bacteria which disseminate throughout blood stream.
      1. Suppurative pelvic thrombophlebitis
      2. Suppurative pyelphlebitis
      3. Suppurative jugular thrombophlebitis
      4. Cavernous sinus thrombosis
   (e) **Thrombophlebitis in varicose veins**—localized inflammatory type—sterile lesions in these veins
   (f) **Ischemic thrombophlebitis**—secondary to acute arterial occlusion or advanced chronic occlusive arterial disease in which there is extensive secondary thrombosis of veins

II. **Secondary complicating thrombophlebitis**—appears to be initiated by rest in bed—prevented by inhibition of clothing mechanism
   (a) **Post-partum thrombophlebitis** ("phlegmasia alba dolens"—"milk-leg") A type of ilio-femoral thrombophlebitis, a lymphangitis secondary to pelvic cellulitis
   (b) **Post-operative thrombophlebitis** — "phlebothrombosis" and inflammatory thrombophlebitis occur 0.05—10.5% after abdominal operations 1.25—12.5% after gynecologic operations — (by various authors) Clot forms first and secondary phlebitis of vein occurs

Site of Clinically Diagnosed Post-Operative Thrombophlebitis

Iliofemoral	254
Short saphenous and deep veins of calf	486
Long saphenous	155
Superficial veins and small varices of leg	14
Veins of arm	7
Other veins	23
Total	939

   (c) **Thrombophlebitis as a late complication of severe injury**—Occurs several days or weeks after injury—Analogous to post-operative thrombophlebitis
   (d) **Thrombophlebitis complicating infectious disease**—pneumonia, typhoid, influenza, acute tonsillitis, pharyngitis, Tb, chronic ulcerative colitis, pyelonephritis, appendicitis, acute infectious arthritis, exanthemata, etc.
   (e) **Thrombophlebitis complicating blood dyscrasias**—polycythemia vera, chlorotic anemias, myelogenous and lymphatic leukemia
   (f) **Thrombophlebitis complicating pregnancy**—rare—may be due to increased venous pressure and slowing of venous blood flow.
   (g) **Thrombophlebitis complicating non-infectious systemic disease**—seen in carcinoma and congestive failure in late stages due to venous stasis.

III. **Primary thrombophlebitis**—no known cause
   (a) **Recurrent idiopathic thrombophlebitis** (Thrombophlebitis migrans)—Seen in large veins of legs, mesenteric, pulmonary, splenic veins—appears to jump from one vein to another—Average age 40 years, 88% men
   (b) **Idiopathic thrombophlebitis of non-recurring type**—Single episode in large vein of otherwise healthy adult
   (c) **Thrombophlebitis associated with thromboangitis obliterans**—Occurs in 40% at some time in course of this disease.

As has been noted, phlebothrombosis is classified as a type of postoperative thrombophlebitis. This is a type, usually asymptomatic, in which the clot is loosely attached to the vein wall, is quite friable, and is composed mainly of fibrin with some cellular elements. There is, therefore, a greater predisposition to becoming detached, producing infarction or fatal embolism. It is believed by some that this type rarely remains a simple process wherein a clot is present within the lumen of a vein, but that in several hours a secondary phlebitis developes as a reaction to the thrombus. It may be, therefore, that "phlebothrombosis" as defined by Ochsner and DeBakey exists as such for only a short time, and then actually becomes a thrombophlebitis.

There is considerable clinical and pathological evidence that phlebothrombosis usually occurs in more than one episode. If the thrombus developes in a small vein, part or all of it may become detached soon after its formation to become a small non-fatal embolus. If it is not detached, or only part of it becomes detached, the clinical signs and symptoms of thrombophlebitis will develope in that particular vein. A second episode may

occur in which the thrombus forms in a larger, more proximal vein, and this clot can become detached to produce a larger or fatal pulmonary embolus. If the first episode occurs in a large vein, such as the external iliac or femoral, the first and only sign may be a fatal pulmonary embolus. It has been noted clinically that a non-fatal pulmonary embolism may precede the clinical evidence of thrombophlebitis by one to three days, or occasionally, as long as a week. Thrombosis may occur in only one leg, or both legs simultaneously, or first one leg then the other, or in veins in other parts of the body. Pulmonary embolism may occasionally be seen in association with the local or primary types of thrombophlebitis, but is seen more frequently in the secondary types, and more particularly, in the post-operative variety.

It is, then, evident that postoperative venous thrombosis is a special type as pointed out by Ochsner, and at least at first is a phlebothrombosis. It is characterized by a spontaneous clotting of the blood, the clot being only loosely connected to the vessel wall. The formation of this type of clot is facilitated by a marked increase in platelet agglutination, by an increase in the erythrocyte sedimentation rate (ESR), and changes in the plasma volume. (Neuda) These changes are noted first, as a rule, on the fourth postoperative day, and are thought to be due to entrance of tissue fluids into the blood as a result of trauma from surgical procedures. A physical state of the blood that would favor thrombosis is then produced. This assumed danger of thrombosis can logically be prevented by the use of anticoagulants. However, there are rarely any signs or symptoms of definite enough significance in this type of thrombosis to always allow the clinician to know of its occurrence, and, therefore, institute prophylactic therapy. Only too often the first manifestation of postoperative thromboembolic disease is pulmonary embolism. However, thrombosis after operation is of rather rare occurrence, and embolism is still more rare. Also, not any thrombosis, but a special type, leads to embolism. For that reason attention should be directed to the formation of this type of clot postoperatively, so that as few cases as possible will require active treatment.

From a practical standpoint, prevention consists in the application of a firm, but not constricting, elastic bandage from the sole of the foot to at least the knee, and preferably higher. This theoretically should be applied to all patients, but many surgeons have modified this to include only the following: (1) those with a history of previous thrombophlebitis or pulmonary embolism; (2) those with evidence of varicosities whether symptomatic or not; (3) elderly patients; (4) those with presumptive evidence of early thrombosis postoperatively, i.e., (a) heaviness or flabbiness of lower extremities (b) acceleration of pulse rate without other signs of congestive failure (c) slight low grade fever without signs of infection (d) mild tenderness in calves of one or both legs. These latter signs of thrombosis respond well to the use of an elastic bandage, and, inasmuch as we assume thrombosis has occurred, anticoagulants are given to prevent further thrombosis and extension of the clot.

The reason an elastic bandage is of definite help has been explained by Neuda as follows: the veins of the lower extremity resemble medium sized arteries in that the tunica media contains an increased amount of muscle. The entire vein wall derives its blood supply from the vasa vasorum which penetrate the muscle coat in order to reach the intima. The reason for this thick muscle coat is that the hydrostatic pressure is higher in the lower extremities than elsewhere, and this assists the blood to return to the heart against gravity. However, in the supine position, the pressure is only one-tenth of that in the standing position, so these veins collapse and the muscle fibers compress the vasa vasorum. The oxygen supply is consequently reduced in the vein wall, and particularly the intima, producing anoxic changes in the tissue cells.

Neuda further believes that this is an important factor in the initiation of the clot and influences proper healing of it. Consequently, proper pressure from an elastic bandage will prevent the compression of the vasa vasorum by the muscle layer, and thrombosis can be prevented, or, if already present, can be influenced to heal in a normal manner by means of a firmly attached thrombus which undergoes organization and fibrosis,

and therefore, will not embolize. The superimposed pressure of the elastic bandage causes counterpressure on the muscle layer allowing the vasa vasorum to remain open.

Therefore, it is believed that the trauma of any surgical operation causes tissue fluid to be introduced into the blood that becomes altered antigenically is some way, in a small number of patients, to cause increased platelet agglutination, increased erythrocyte sedimentation rate and alterations in plasma volume. This in itself is not sufficient in all cases to cause thrombosis, but with anoxic changes in the intima due to collapse of the veins, a loosely attached clot may form with its predisposition to embolization.

Further prevention of postoperative thrombosis consists in avoidance of pressure on the legs during operation, avoidance of excessive pressure from tight bandages, dressings or adhesive tape, instituting exercises for the legs and feet which should be started soon after operation, massage to the legs daily, as well as early ambulation.

The management of the patient when definite thrombosis has been known to have occurred consists in early institution of anticoagulant therapy. Heparin should be given immediately, either intravenously in 50 mgm. doses q. 4 hr. or 250 mgm. in oil intramuscularly in daily doses, and continued for at least 36 hours. A blood prothrombin time should be obtained, and dicoumarol given at the same time in an initial dose of 300 mgm. Subsequent doses will depend on the level of the daily prothrombin time, and this should be kept between 30% of normal (27 seconds) and 10% of normal (60 seconds) by the Quick method or one of its modifications. Experience with a large group of cases has shown that thrombosis is inhibited in that range.

Below 10%, bleeding from mucous membranes and beneath the skin may occur. Anticoagulants should not be given until 48 hours postoperatively to allow firm clots to form at the areas of hemostasis. However, the indications for their use are not common until after this period. When the prothrombin time has reached a therapeutic level, heparin is discontinued, and dicoumarol or one of its synthetic derivatives is used alone for a week or ten days when all evidence of thrombosis has disappeared. Besides the use of anticoagulants in patients having demonstrable thrombosis or a non-lethal pulmonary embolus, firm dressings should be applied from the toes to the groin in the form of warm, moist flannel strips. In those with suspected thrombosis, or with minimal signs, an elastic bandage can be applied as already described.

Femoral ligation has recently been practiced by surgeons, and is still advocated by some, when pulmonary infarction or venous thrombosis are present. Several objections to this treatment rather than the use of anticoagulants have become evident during recent years. Bilateral femoral ligation does not prevent embolism from a second episode of thrombosis in the internal iliac veins or in veins other than in the legs. In cases of pulmonary embolism without evidence of thrombophlebitis in the legs, it is not certain that the embolus came from one of the leg veins. The incidence of fatal embolism is only 6% in cases of thrombophlebitis of the short saphenous and veins of the calf, and is quite rare from varicose veins, or the long saphenous vein. It is obvious, then, that many unnecessary operations would be done if bilateral femoral ligation were done in all such cases, and a second major operation is added to a patient who is already in a postoperative state.

Ligation of the inferior vena cava, however, seems to have a place in the therapy of thrombo-embolic disease. In suppurative pelvic thrombophlebitis, we have a condition characterized by the formation of a firm, adherent clot in the affected veins in which there is a greatly lessened incidence of lethal embolism. The clots that are formed are septic and tend more frankly to become purulent. When liquefaction takes place, infected emboli are liberated in showers. Pyemia or septicemia result and the disease often tends to be fatal. The thrombi are almost equally distributed between two primary highways of venous return: (1) the uterine, draining into the hypogastric and common ilian and then to the inferior vena cava; (2) the ovarian, draining directly into the inferior vena cava on the right, and indirectly by way of the left renal on the left. In a recent report of

70 cases of pelvic septic thrombo-phlebitis proven by palpation of the veins or histologic examination of resected ovarian veins, clinical evidence of pulmonary embolism was found in 27 with X-ray confirmation in 24. In four of the 27 cases, lung abscess occurred, requiring lobectomy in 2 and lobotomy in one. In two cases, subacute bacterial endocarditis occurred, and in one, cerebral abscess. In the 8 cases that died, death resulted from pyemia and septicemia, and in none was massive pulmonary infarction present.

The diagnosis is often difficult, but signs and symptoms indicate a septic process and thrombosed veins can be palpated in the vagina and pelvis in 60% of cases. Surgical treatment is indicated in those who fail to respond to adequate medical treatment within 4 or 5 days in whom a pulmonary infarct has occurred. This consists in ligation of the right ovarian vein and inferior vena cava above the clot, but always below the renal vein. The transperitoneal approach is taken if no previous hysterectomy or bilateral salpingoophorectomy has been done. A right lumbar sympathectomy is done at the same time. If the left ovarian vein is also involved, this is ligated above the clot and a left lumbar sympathectomy is done. If previous hysterectomy has been performed, a right extraperitoneal approach is taken, the vena cava ligated and a right lumbar sympathectomy done.

Other indications for inferior vena cava ligation are present in those patients in whom future extensive surgery is anticipated, and who have already suffered a recent embolism, and in those cases where thromboembolism cannot be controlled by the usual anticoagulant therapy because of some contraindication to its use. Foley, the urologist, has recently used this procedure as a prophylaxis against pulmonary embolism in the coagulation diathermy treatment of infiltrating bladder tumors. In these cases, the right extraperitoneal approach is used.

Another disease for which active surgical treatment is advocated, at the present time, is arterial embolism. This results from mural thrombi as a result of auricular fibrillation or myocardial infarction, from thrombosis at the site of an atheromatous plaque, or aneurysm of the aorta. This is not a common phenomenon, fortunately, and no one surgeon has had a large experience with its surgical treatment. Patients suffering from this condition are usually poor surgical risks because many are of advanced age and most have severe cardiac disease or other serious conditions. The emboli lodge at the bifurcation of vessels, as a rule, and, consequently, they will be found at the bifurcation of the aorta, common iliacs, or common femoral arteries. The signs indicating their presence, i. e., sensory, motor, and temperature changes will be below the site of embolus.

The role of the surgeon is to localize the embolus and remove it by means of an incision in the affected artery at this point. Bleeding is controlled proximally and distally by artery clamps or the application of soft rubber tubes. Heparin is administered intravenously before, during, and after the operation in sufficient dosage to keep the coagulation time at 15 minutes. Arteriectomy, removing the segment containing the embolus, may be necessary if embolectomy is not possible due to its adherence to the intima.

The results from this procedure may be considered disappointing when it is realized that the hospital mortality is high, and that eventual amputation after peripheral embolectomy is not infrequently necessary. The best results can be expected if the duration of the clot is less than eight hours; if the site of the obstruction is at the bifurcation of a large vessel; if a normal arterial wall is present at the site of the embolus; if there is an absence of multiple emboli; and if the patients are relatively young, particularly those with auricular fibrillation.

REFERENCES

Early Diagnosis and Prevention of Postoperative Thrombo-embolism, Neuda, P. M.: Amer. Jour. Surg. 84: 627-635; Dec. 1952.

Suppurative Pelvic Thrombophlebitis—I. Pathology and Etiology, Collins, Conrad G., MacCallum, Evan A., Nelson, Edward W., Weinstein, B. Bernard and Collins, Jason H.: Surgery, 30: 298; 1951.

Suppurative Pelvic Thrombophlebitis—II. Symptoms and Diagnosis. Idem.; p. 311.

Suppurative Pelvic Thrombophlebitis—III. Surgical Treatment. Idem.; p. 319.

Femoral and Iliac Embolectomy, Klingensmith, W. and Theis, F. V., JAMA, 150: 1393-1395 (Dec.) 1952.

Peripheral Vascular Disease, Allen, E. V., Barker, N. W. and Hines, E. A. Philadelphia: W. B. Saunders & Company, 1946.

THE EARLY DIAGNOSIS OF POLIOMYELITIS

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Acute poliomyelitis is a systemic disease of virus etiology. The various strains of viruses thus far identified have a predilection for the central nervous system and the voluntary muscular system. The disease is essentially a medical problem and competent and adequate medical management is a prerequisite to obtain results therapeutically profitable to the patient. This medical management must be augmented by conscientious nursing care, high protein diet, and intelligent use of physiotherapy directed to the pathologic physiology in the muscles.

The main reliance for diagnosis is placed upon complete physical and neurological examination, which must be made as soon as possible.

It is generally understood that during poliomyelitis epidemics 80 per cent or less of those who are susceptible to poliomyelitis may develop the abortive type of the disease in which the symptoms may be transient sore throat, nausea, headache, and listlessness. These symptoms clear up entirely within 5 to 7 days so that these patients are rarely diagnosed or reported as poliomyelitis. It is only in research projects that the virus has been isolated from the nose and throat washings or from the feces of such patients and the spinal fluids have been found positive.

Twenty per cent or more of those who are susceptible in any outbreak will have one of the several types of reportable poliomyelitis. Fever is usually present. This may subside in 2 or 3 days and then reappear, or it may appear together with the symptoms of pain and stiffness in the muscles of the limbs, back, or neck. The temperature usually drops in 5 to 7 days, but the other symptoms continue and such a patient is diagnosed as a nonparalytic poliomyelitis and kept under isolation according to prevailing public health regulations. Spinal fluid findings of pleocytosis, increased total protein and positive globulin will confirm the diagnosis. Positive spinal fluid findings help to confirm the diagnosis, but negative findings are of no significance in the presence of clinical signs and symptoms.

This patient must now be watched carefully for any paralysis or bulbar symptoms, which may appear any time from the second to the eighth day after the onset of the symptoms. The early symptoms of respiratory and circulatory failure must be appreciated and treated immediately, if bulbar cases are to be saved. The severity of the paralysis cannot be predicted. As a rule, no extension of the paralysis is seen after the eighth day, but failure of the fever to subside will usually means extension of the disease. Patients must be observed several times a day during the acute, spreading phase. This is done best with the patient in a hospital which is staffed with experienced personnel and which is equipped with oxygen tents, oxymeters, electrical suction machines, resuscitators, respirators, spirometers, rocking beds, intermittent positive pressure machines, electrophrenic respirators, an electromyograph machine, tracheotomy and bronchoscopy facilities, rehabilitation apparatus as wheel chairs and walkers, hot pack machines, tubs or a Hubbard tank for hot baths, and facilities for orthopedic appliances.

The signs of bulbar poliomyelitis are those presented by partial or complete involvement of any of the cranial nerve nuclei. The cranial nerves and their functions are:

I. Olfactory—Smell

II. Optic—Sight

III. Oculomotor—Eyeball muscles except IV and VI; upper lid, constrictor of pupil and muscles (ciliary) of accommodation. Paralysis causes ptosis of the eyelid; eye deviates out and downward; diplopia; pupil is fixed to light and accommodation.

IV. Trochlear—Superior oblique moves eye down and out.

V. Trigeminal—Muscles of mastication. Sensation to face and mucous membrane of conjunctiva, nose, mouth, tongue and uvula. Also taste fibers to anterior two-thirds of tongue.

VI. Abducens—External rectus moves eye outward.

VII Facial—Facial muscles except those supplied by the V. Paralysis causes inability

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to wrinkle forehead, to close eye, to whistle, or to blow out the cheeks.

VIII Acoustic—Hearing and equilibrium. Paralysis causes tinnitus and nystagmus.

IX Glossopharyngeal—Supplies gag reflex, and taste to the posterior third of the tongue.

X Vagus—Supplies heart, lungs, respiratory system, and all abdominal organs except pelvis. Controls speech, swallowing, and soft palate.

XI Spinal Accessory—Supplies trapezius and strenomastoid. If paralyzed, patient cannot shrug shoulders or lift head from supine position.

XII Hypoglossal—Muscles of the tongue. Tongue deviates to the paralyzed side.

An experienced nurse must be constantly on the watch for such signs as rapid or irregular pulse, hypertension, restlessness, shock, dyspnea, cyanosis, anxiety, apprehension, shallow or irregular respirations, nasal speech, inability to swallow, severe drowsiness, regurgitation of fluids through the nose, twitching of muscles, or the use of neck muscles in inspiration. Paralysis of the upper extremity may be the beginning of paralysis of the diaphragm on the same side through the involvement of the phrenic nerve. Loss of the abdominal reflexes may mean paralysis of the lower six thoracic segments of the spinal cord and paralysis of the abdominal muscles which are of major importance in respiration.

The differential diagnosis must be made carefully as soon as the patient is seen. There are many acute illnesses which simulate poliomyelitis. About 50 different diseases were diagnosed in patients admitted for poliomyelitis in our clinic during the past 12 years. This disease is most frequently misdiagnosed as "summer grippe."

The following table summarizes the signs which, when present, usually exclude poliomyelitis. The disease which usually simulate this disease are also listed.

Signs Which Usually Exclude Poliomyelitis

Signs	Commonly Confused Conditions
1. Rash of any kind	Meningococcic meningitis and petechia; Rocky Mountain spotted fever.
2. Swollen glands	Infectious mononucleosis, neuroblastoma, mumps meningoencephalitis.
3. Swollen joints	Acute arthritis, rheumatic fever.
4. Localized joint or bone tenderness	Bone tumors, osteomyelitis, rickets, scurvy, tuberculosis, epiphysitis, cervical or lumbar disc.
5. Bleeding gums	Blood dyscrasias, scurvy.
6. Unilateral or bilateral eyeground changes	Brain tumor or abscess.
7. Positive blood or spinal fluid serology	Congenital or acquired lues.
8. Purulent spinal fluid	Meningitis—influenzal, meningococcic, pneumococcic, staphylococcic, streptococcic.
9. Hemorrhagic or xanthochromic spinal fluid	Cerebral accident, tumor in brain or spinal cord.
10. Convulsions	Encephalitis, epilepsy, meningitis, mumps, spastic paraplegia, tuberculosis, cerebral thrombosis.
11. Extreme drowsiness without muscle spasm or muscle paralysis	Coma-diabetic, uremic, emcephalitic.
12. Sensory changes to heat, cold, pain, touch, vibration, or position	Arachnoiditis, brain tumor, hysteria, nucleus pulposus, peripheral neuritis, peroneal palsy due to syphilis, spinal cord tumor, syringomyelia, transverse myelitis, multiple sclerosis.
13. Extreme weakness without muscle paralysis	Muscular dystrophy, myasthenia gravis, progressive muscular atrophy.
14. Septic temperature	Influenza, pelvic inflammatory disease, pneumonia, septicemia, Still's disease, subacute bacterial endocarditis, leukemia, typhoid fever.
15. Paralysis of soft palate and muscles of accommodation	Myasthenia gravis, post diphtheritic paralysis.
16. Jaundice	Acute infectious hepatitis, Weil's disease.
17. Other conditions confused with poliomyelitis	Appendicitis, aspetic meningitis, Bell's palsy, laryngotracheo-bronchitis, lymphocytic choriomeningitis, New Castle disease.

SUMMARY

Clinically, poliomyelitis may be abortive, non-paralytic, or paralytic. The paralytic types may be spinal, bulbar, or encephalytic in the order of greatest frequency. The characteristic finding in the non-paralyzed patients is muscle shortening due to muscle rigidity or "spasm"; this is more prominent in the entire posterior area of the body. This muscle contraction is not necessarily related to any paralysis of any of the "opposing" muscles groups. In the paralytic patients the characteristic finding is, in addition to the muscle shortening noted in the non-paralytic, a flaccid paralysis of muscle groups, asymmetrically located.

Recovery is spontaneous and complete only in the abortive type. The other types require special muscle relaxation so that a full range of motion of all joints is obtained and maintained, special muscle exercises, special nutrition, and controlled prolonged medical observation in order to obtain maximum recovery and maximum use of the neuromuscular power the patient still retains. The amount of special care a patient will require will depend on the severity of the involvement.

The mortality rate is generally influenced by the previous experience of the locality-early diagnosis, early hospitalization, and the experience of the hospital personnel. For these reasons it is always higher in an area where an outbreak appears for the first time. Delaware has had several outbreaks of poliomyelitis—in 1944, 1947, 1948, 1952.

THE DELAWARE ACADEMY OF MEDICINE

Past, Present, and Future

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Wilmington, Del.

As a background for tonight's discussion, some interesting educational and medical dates in Delaware seem appropriate.

1776—On February 3 the Medical Society of Delaware was incorporated under the name "The President and Fellows of the Medical Society of Delaware." First president was Doctor James Tilton. The Tilton home was on the property which is at present the University Club, at Ninth and Broom Streets. Founded in 1776, the Medical Society of Delaware is the second oldest State Medical Society in the country: the first was New Jersey, founded 1766.

1843—The University of Delaware was founded as Newark Academy.

1888—The Memorial Hospital was founded The Wilmington Homeopathic Hospital.

1889—Delaware State Hospital at Farnhurst.

1890—The Delaware Hospital in Wilmington was founded.

1907—Milford Memorial Hospital in Milford, Delaware.

1910—The Wilmington General Hospital, at first located at Eighth and Adams Streets.

1916—Beebe Hospital in Lewes.

1924—St. Francis Hospital in Wilmington.

1927—The Kent General Hospital in Dover.

Prominent among Chiefs of Service in 1928 in the Wilmington hospitals were such names as James Draper, George Forrest, Paul Lukens, McEntee, John Palmer, Henry Ward Briggs, Frank Pierson, Albert Robin, Willard Springer, and Joseph P. Wales. There were no residencies in any of the hospitals and, indeed, only a few interns. At the Delaware Hospital there were perhaps four or five interns; at the Memorial Hospital two or three; and one or two at the Wilmington General, and the St. Francis Hospital was just starting. There was not a medical library in the entire state of Delaware. No one individual had more than the bare necessities of a working library of his own. For any reference work it was necessary to go to Philadelphia or Baltimore.

In such a state of affairs, the need for a medical center and a medical library seemed urgent to the few individuals who later became the founders of the Delaware Academy of Medicine. According to the history of the Academy as written by Dr. William H. Kraemer for its tenth anniversary celebration May 9, 1940, the founders first met in Dr. Flinn's office at 503 Delaware Avenue on December 12, 1929. Numerous meetings were held after that mainly in the Y. M. C. A. over the next several months and the next year. With the onset of the great national economic depression in 1930, the likelihood of the establishment of an Academy of Medicine with a building of its own seemed very remote. However, early in 1931 Mrs. Henry B. Thompson and others became interested in saving the old Delaware Bank Building at Sixth and Market Street which was about to be razed. That building was completed in 1816 and in 1865 became the National Bank of Delaware, the fourth oldest National Bank in the country. With this history, and because it was a particularly good example of colonial architecture, citizens in the community were anxious to save it. They therefore were very glad for it to be used as the home of the Delaware Academy of Medicine. In spite of the depression, $36,000 was collected and the cornerstone was laid on June 1, 1932. With the exception of a few alterations for modern use, the building was erected on its present site exactly as it stood in 1816. The original hand-hewn rafters joined with wooden pegs, the staircase, floors and trim and the original iron railing surrounding the building have all been preserved. The site was purchased from Joseph Bancroft and Sons Company. This property can be traced back through the Lov-

*Abstract of an Address given at the Delaware Academy of Medicine at the Annual Meeting, Tuesday, January 27, 1953.

erings and the Shallcrosses to Thomas Gilpin, Jr., who, on the "Sixth day of the Sixth month in the Sixth year of the reign of our Sovereign, George the Third over Great Britain, etc., King, etc., and in the year of our Lord 1766" (June 6, 1766) received the land by virtue of a patent from the commissioner of property under the Honorable Thomas Penn and Richard Penn, proprietors of the Counties of New Castle, Kent and Sussex on Delaware and the Province of Pennsylvania. Thomas and Richard Penn were the sons of William Penn and his second wife, Hannah Callowhill. The tract patented to Gilpin was of one hundred eighty acres and extended southerly from the Brandywine Creek and northerly from the Rattlesnake Run.

The cornerstone laying and dedication of the building took place on June 1, 1932. The cornerstone was laid by Mayor Frank C. Sparks after the deed to the property had been presented by Mrs. Thompson to Dr. Lewis B. Flinn, then President of the Academy.

Mrs. Thompson told the interest shown in preserving the bank structure and how some people contended that it would be impossible to raise funds then during the depression. However, at that time, about $33,000 had been raised.

The ceremonies were opened with an invocation by the Reverend Dr. Charles F. Penniman, then rector of Trinity Episcopal Parish, and closed with a benediction by the Rev. J. Francis Tucker, at that time pastor of St. Anthony's Church. Dr. William H. Kraemer presided.

Prior to the laying of the cornerstone, the deed and other articles given by Mrs. Thompson were placed in a copper box, together with a roster of the Delaware State Medical and Dental Societies, a copy of the original resolutions of the Delaware Academy of Medicine, copies of medical journals, currency of that date and clippings from recent issues of the local papers telling of plans for the cornerstone laying.

The Academy, however, was in a very precarious financial status in 1933 and 1934 because of an outstanding debt of $12,000 for reconstruction expense. It is felt that had not a third group been formed to underwrite this $12,000 the whole project might have been lost. It may be of interest to again record the names of those individuals who formed this holding company—Mrs. Henry B. Thompson, Mr. Henry B. Thompson, Mr. Daniel F. Shields, Jr., Dr. Lewis B. Flinn, Mrs. Ernest duPont, Dr. Victor D. Washburn, Allen L. Lauritson Company, Mr. Thomas Donaldson, Dr. Charles P. White, Dr. Charles L. Reese, Dr. Frank L. Grier, Mr. John T. Chandler, Jr., Dr. William H. Kraemer.

The founders of the Academy were: Drs. Julian Adair, O. S. Allen, J. M. Barsky, W. E. Bird, J. D. Brown, L. B. Flinn, G. W. K. Forrest, W. H. Kraemer, W. O. LaMotte, E. R. Mayerberg, J. H. Mullin, W. M. Pierson, J. G. Spackman, A. J. Strikol, C. E. Wagner, and V. D. Washburn.

A librarian, Mrs. Ava Taylor Watson, was engaged September 1, 1933. Mrs. Watson loved the Academy of Medicine and spent great thought and energy in its behalf. She was always suggesting things of interest for the members and trying to facilitate use of the library and doing her very best to insure that the Academy carried forward its purposes and functions. Her untimely and sudden death in 1938 was a great loss to this institution. Upon Mrs. Watson's death, Mrs. Edward Mendenhall very kindly stepped into the breach and acted as librarian for a period of three years. After the onset of World War II which, incidentally, took into military service over 35 per cent of the Academy's membership, Mrs. Gerald Beatty acted as part-time librarian during the war years and was of tremendous aid in making it possible for the institution to weather the difficulties of the war. In spite of the sudden decrease in membership and necessary decrease in activities in use of the Academy, some progress was made even during this period. Mrs. Beatty continued for a short time after the war until the present custodian, Mr. Syrovatka, arrived. The latter had had considerable banking, legal, and political experience in Prague, various European countries and Mexico, and found himself finally in this country. Our government thought well enough of him to have a special bill

passed through Congress to grant him asylum here and give him the opportunity to acquire citizenship. He has been of great service to the Academy and has made many valuable and constructive suggestions.

The list of presidents of the Academy is as follows: Dr. L. B. Flinn, 1930-1941; Dr. William Kraemer, 1941-1945; Dr. Edgar R. Miller, 1946-1947; Dr. G. A. Beatty, 1948-1949; Dr. B. M. Allen, 1950-September 1951; Dr. W. M. Pierson, September 1951-January 1952; Dr. L. C. McGee, 1952-1953.

It may be of interest to note that in the president's report of November 1936 the annual income totaled $3500, with $1650 from members, $450 from the Endowment Fund, and special contributions $1400. At that time the librarian's salary was $900, there were about 120 members, and 120 current periodicals. Contrast this with the president's report at the present meeting which indicates $3,000 from membership dues, $5,000 from contributions from associate members, $330 only additional contributions by the full active members, income from the Endowment Fund or Trust Fund $1369, and contributions for the use of the building $1670, total receipts of $12,000.

During the past year, sixteen new members have joined the Academy. The total professional membership is now 220, the number of associate membership is 125. The library has acquired 52 new volumes and 11 new periodicals during the current year and the use of the library by the members has increased 20 per cent during the past year. The president's report also indicates that there has been considerable activity in the form of courses, lectures to the public, and special scientific meetings conducted by the Academy.

What can we legitimately foresee as to the future of the Delaware Academy of Medicine? First, let us go back to the original Certificate of Incorporation. From this document I would like to quote three sentences:

1. The purpose of the Academy is "to foster among its members interest in medical, scientific, literary, and educational conditions and to render service without recompense towards these ends in the State of Delaware."

2. The Academy was organized "solely for the purpose of rendering service without recompense and for charity to scientific, literary and educational ends."

3. "To aid, cooperate with, or endow any individual, association or corporation engaged in charitable or educational work within the State of Delaware; to collect statistics and information and to publish and distribute documents, reports and periodicals to carry on such educational and charitable work along the lines of its corporate purposes as it may deem wise; to provide for and furnish public instruction in hygiene, sanitation, and the laws of health; to conduct lectures and meetings, to acquire and maintain a library and in general to do and perform all things necessary or convenient for the objects of the corporation or any of them."

I suggest, therefore, that the functions of the Academy are four: (1) a meeting place; (2) a library; (3) education; (4) research. Let us examine each category briefly and suggest how best we may improve and develop the function of each. The Academy is naturally a meeting place for regular and special meetings of the Academy. It is also a meeting place for some smaller groups. A Journal Club, for instance, has been meeting regularly once a month at the Academy ever since the war. Prior to the war, a number of sectional meetings were held, particularly a section on ophthalmology, another on obstetrics and gynecology. At that time there were very few sectional meetings held in the hospitals. The development of the residency program and a large number of departmental meetings and conferences has largely consumed the time and interest of the specialty groups so that sectional meetings in the Academy have not been renewed. Perhaps the Academy, as part of its educational program, should work more closely with these various special groups to give library service and other educational features as may be practical from time to time. Meetings have also been held at the Academy for the public and other interested groups on medical and related matters. The auditorium, at times, is very nearly full with some of the regular meetings of the regular medical organizations, such as the New Castle County

Medical Society. The question has arisen as to whether the auditorium should be enlarged or another large auditorium added to the north side of the building to accommodate larger and special medical meetings which currently have to be held elsewhere. This matter needs further consideration.

The function of the library can properly be divided into three—the reading room, an extern service, and a cooperative library plan. The reading room includes service of a librarian. The difficulty arises in the membership having insufficient time to spend in the reading room during library hours. Previously, keeping the library open one or two evenings a week has been explored but it has not been found worthwhile. The librarian is, however, available over the telephone and will very gladly look up certain requested literature or journals and have the material available in one pile so that the member may call and either read this matter in the reading room or take it away according to the rules laid down by the library committee.

I feel very strongly that the extern service should be developed and brought to the attention of all members. If, by writing or by telephone to the librarian, certain journals or reference books are requested, the Academy should make arrangements for this material to be sent by messenger to the member's office, home, or hospital if it is within close range; otherwise by mail. If this type of service were improved so that a member could have on his desk within a few hours or certainly by the next day certain literature in which he was interested at the moment, the Academy would be doing a great service and I am sure the library would be found of much greater use than it has been heretofore.

In line with this so-called extern service, a cooperative plan with other libraries in the area should be developed. We have nearby many special libraries particularly having to do with chemistry or physics or other special branches; for instance, at the University of Delaware, at the Hercules Powder Company Library, at several special libraries of the DuPont Company, the Alfred I. duPont Institute, etc. It is not necessary or desirable that the Academy should attempt to have available all the reference work which is covered by all these institutions. Many of the latter use our library regularly. We have here much literature which the other organizations do not carry. We are glad to have them use our facilities. Frequently we have need of their facilities. It would be better, however, if this cooperative effort were set up with more smoothly working mechanics than has been established up to now.

In addition to promoting education through the facilities of the library itself, the Academy has a function to perform in stimulating and directing education more directly either by conducting courses, giving lectures, panel discussions, forums (both professional and for the laity) or in cooperating with other institutions in educational activities. It is in this latter category where I feel we can make our greatest improvement. We must endeavor to keep in closer touch with the hospitals. The cooperative library plan can be developed here with considerable advantage. Also, we should keep in touch with the various educational programs having to do with post-graduate education as well as with stated hospital meetings, help prevent overlapping of conferences if possible and make available to the hospitals any particular educational advantages which we may have or may acquire. We also should develop closer association with special organizations such as the American Heart Association, American Cancer Society, American Tuberculosis Association, American Diabetes Association, etc. In addition, and in keeping with the original Certificate of Incorporation, the original Charter of the Academy, one of our functions is to take an active part in public health—not in its administration but in the educational sphere. We should, I think, make a more direct approach both to the State Department of Health and the Wilmington City Board of Health. We have a responsibility here which we have not fulfilled.

Lastly, we have the question of research. It certainly is not the function of the Academy of Medicine actually to engage in medical research. We have not and have no rea-

(Concluded on Page 126)

+ Editorials +

DELAWARE STATE MEDICAL JOURNAL

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AMA Convention

The AMA Convention will be held in New York, June 1-5. So far as we have been able to ascertain, no Delaware doctor has yet received his membership card, which is used at the time one registers at the Convention. The AMA reports that membership dues for 1953 are now rolling in at a much faster rate than in 1952. The headquarters office is equipped to mail out about 10,000 cards a week, but since the dues are coming in at one time and in such a big volume there is quite a backlog already on hand. To make matters worse, there are five states that have not yet sent in any of their dues to the AMA, whereas certain other states, notably Colorado, Massachusetts, Ohio, Pennsylvania and Texas have already mailed in more than 80% of their membership dues. Even in little Delaware one County Society, as late as April 27, had not yet sent in any dues to our State Treasurer. These circumstances might present quite a problem when it comes to registering at the Convention. We know of two ways by which this handicap may be averted.

1. The doctor should send in *immediately* to the AMA the Advance Registration Blank, which has been printed in JAMA every couple of weeks from early in February. Should Chicago not be able to send back to him the part that he is to use in New York, we can fall back on the second remedy.

2. As our State Treasurer receives the dues from the County Treasurers he will mail to each physician a Temporary Receipt Card, a supply of which has been ordered from Chicago. It may take another ten days for these Temporary Receipt Cards to be received by our Treasurer, but as soon as he gets them he will mail them out to our members. At best the interval between the receipt of these cards and the Convention itself is quite short, so it is urgent for everyone to attend to these matters promptly.

Chicago has a complete roster of our members up to this date, which should eliminate the possibility of a Delaware doctor being refused registration. None-the-less this 1953 jam demonstrates the importance of paying dues promptly in January, of the County Treasurers remitting to the State Treasurer in February, so that he can remit to the AMA also in February. Even then the AMA would be pushed to the utmost to get out 140,000 Membership Cards in time for a June Convention.

Bad Publicity

The thousands of our citizens who listen to Joe Pyne's program, "It's Your Nickel" on WILM were treated, around March 20th, to some news about the medical profession that was definitely hurtful to us. A woman called in and told Mr. Pyne that she had received a bill from a local doctor for $20 for a broken appointment. She explained that she broke

the appointment because her child was sick, and she asked Mr. Fyne what to do about it. He said to do nothing about it, and that no doctor would sue for a mere $20. Following this dialogue there were numerous calls expressing disgust at a profession that would resort to such tactics. One caller said he was not a lawyer but was a law student and that the doctor could not collect that bill. Another disgusted citizen said that he was not a physician but he knew the doctors had a Grievance Committee to consider just such complaints from the public. Several irate callers said such tactics, if persisted in, would bring about a demand for Socialized or State Medicine. And other comments equally uncomplimentary to the medical profession. Incidentally, the lady later engaged legal counsel, who was ready to go to bat for her.

About ten days later this woman called Mr. Pyne again and stated that she had received a letter from a collection agency to the effect that if this $20 bill was not paid within three days the matter would be taken to court. Again the telephones jangled with calls of criticism of our doctors pouring into the ears of Mr. Pyne, who repeats them on the air, phrase by phrase. Be it said to the credit of this gentleman he discounted most of the complaints and stated that his impression of the medical profession here was a distinctly favorable one, and that the doctor in point must be the rare exception.

This patient called Mr. Pyne for a third time about two weeks later and stated that she had consulted the chairman of the Grievance Committee of the Medical Society and that he had told her to forget the whole thing. Thus, apparently, ends the publicity in this particular instance, but upon the untold thousands of people who heard it a bad impression has been imposed, an impression that we all know is unjustified. Of course the episode was the rare exception. We mention this matter only because one-half the town already knows about it, and we would like to give the public the knowledge that such an episode will probably never occur again in Wilmington.

THE DELAWARE ACADEMY OF MEDICINE

(Concluded from Page 124)

son to expect facilities for doing this either in the form of laboratories or in necessary funds. We should, however, and in fact it is our responsibility, stimulate and encourage medical research in this area, knowing that such research properly conducted will add immeasurably to the caliber of medicine practiced in the community. We must be very careful, however, before approving any particular investigative effort that the individuals concerned and the project under consideration will pass critical authoritative examination. The function of the Academy of Medicine through its Research Committee is to make certain that any research matters brought before it for an opinion will receive competent inspection and examination.

If the Board of Directors and the Executive Committee will see to it that some individual or individuals will constantly direct and stimulate and encourage activity in these four main fields of function, the Delaware Academy of Medicine can go far in the foreseeable future toward fulfilling the purposes laid down in its charter. If this is done, and I believe it can be done, a great service will be rendered to both the professional and non-professional members of this fast growing community.

Our opportunity is great: it is ever so in the field of medicine. From a lecture by Benjamin Rush, delivered November 7, 1791, I quote: "We live, gentlemen, in a revolutionary age. Our science has caught the spirit of the times, and more improvements have been made in all its branches within the last twenty years, than had been made in a century before."

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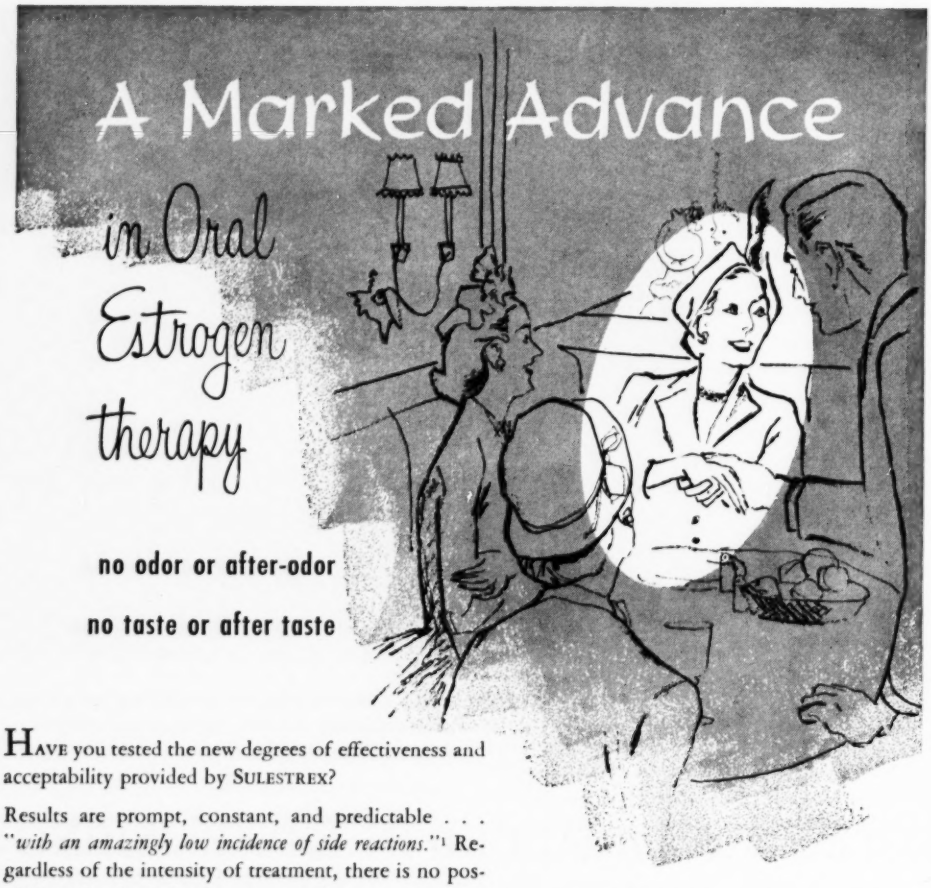

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1. Reich, W. J. et al. (1952), A Recent Advance in Estrogenic Therapy. II. Amer. J. Obst. & Gynec., 64:174, July.

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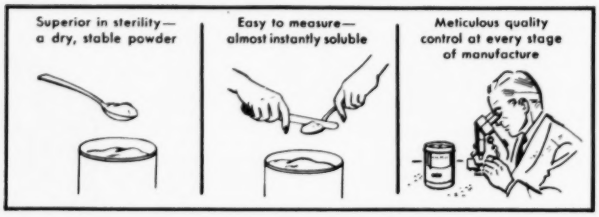
A formula supplying 20 calories per fluid ounce is easily prepared with 1 part evaporated milk and 2 parts water, plus 1 tablespoon Dextri-Maltose to each 5 ounces



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